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EDWARD ALBERT WOODWARD.[1]

By R. E. LONGWORTH,
Lismore.

IN presenting this memorial on the life of the late Dr. E. A. Woodward, I am conscious of the great honour the Federation of Country Local Associations has entrusted to me. The responsibility of the task of adequately paying this tribute has been something of an ordeal to me, which I trust will not be transferred to you in the hearing.

Edward Albert Woodward was a man beloved by those privileged to know him personally or professionally. Truly of him could be said: "A man he was to all the country dear."

Amongst country practitioners it has been my experience, particularly in this north-east corner, to observe the community spirit and cooperation which exist in our ranks. Some of our problems are basically different from those of our metropolitan confrères, and their discussion and disposal frequently demand much time and travel by members in attending our meetings.

At such meetings it has been customary to interpose short social breaks in the business. This has enabled members, as well as their wives, to become well acquainted and has contributed in no small degree to that strength and harmony so essential in local medical associations. It must follow that unity of purpose and smooth and efficient functioning of country local associations are a *sine qua non* for the successful achievement of FOCLA's objectives.

In E. A. Woodward, while health permitted, we had a staunch supporter of the North-Eastern Medical Association, and it is to be regretted that he was not with us to see the growth of FOCLA.

Medicine, like any avocation, has a personnel comprising many varieties of disposition, capabilities, educational attainments and recreations. It is not uncommon to find practitioners, accomplished in their profession, who have not time or inclination for exploring those fields beyond the medical horizon. Two of my friends, one of whom became an eminent Macquarie Street specialist, told me that beyond the daily Press, medical literature and reading prescribed for their examinations, they had done no reading at all.

In any distinguished company, a man of E. A. Woodward's calibre would be outstanding, not only for the diversity of his interests, but also for the degree of excellence to which he attained in all of them.

To achieve distinction as a classical scholar, physician and pathologist, citation for outstanding military service, and prominence in many forms of sport, is a remarkable record. The writing and translating of Latin and Greek

[1] A memorial oration to the late Dr. Edward Albert Woodward, delivered at the annual post-graduate course of FOCLA, New South Wales, on August 13 to 17, 1956, at Lismore.

verse as a relaxation remained all his life, and he enjoyed reading in French, particularly verse. The late W. A. Purves, Headmaster of Sydney Church of England Grammar School, considered that, apart from the original Greek masters, E. A. Woodward's verses were the most beautiful he had read.

One frequently observes that many gifted persons, who have developed their talents to the full, show little patience with those of lesser mental calibre. To the latter, E. A. Woodward presented a genuine spirit of tolerance even when their views differed from his own. When one finds these gifts almost concealed by a modesty innate and unassumed, there seems the complete answer to the question: "What is a gentleman?" A delightful sense of humour, untinged by malice, completed the picture. I have never heard him refer to any of his feats, unless it was in association with an incident in which his team or crew took the major part of the stage. Add to his devotion to his work as a country practitioner the virtues I have enumerated, and the commemoration of his life appears a logical choice for reflection by our assembly today.

My personal, professional and army friendship with Woodward extended over many years, but for many of the facts preceding that time I am indebted to his wife, son and daughter, to his brother, Lieutenant-General Eric Woodward, C.B., D.S.O., G.O.C. Eastern Command, and last, but not least, to Dr. Eric Holland and Dr. N. St. C. Mulhearn, two of his partners in group practice.

Edward Albert Woodward was born in Melbourne. His early boyhood was spent in the Hay district of New South Wales, where his family had a grazing property. Later they moved to the Charleville district and subsequently to the Darling Downs, where his father managed other pastoral holdings till his death, which occurred while his son was still a schoolboy.

His early schooling was received at the Toowoomba Grammar School. Here he gave promise of the bright academic years which were to follow, and in 1910 he won the University of Sydney medal for first place in Greek in the Junior Examination. His subsequent education was continued at the Sydney Church of England Grammar School, and in the Senior Matriculation Examinations of 1912 he gained first place in English, Latin and Greek, and the University medals. He had also been prominent in the sporting side of both schools. Although he was awarded the Cooper classical scholarship he by-passed this —a decision entailing a sacrifice of inclination to duty; he felt that its acceptance would lengthen the period of dependence on his widowed mother, who was sparing no effort to ensure the best possible education for her two sons, and so he proceeded to the Faculty of Medicine.

His remarkable capacity for application to a diversity of studies and interests reasserted itself during his early student years. This characteristic was to remain till the close of his life. For a student, in addition to obtaining excellent results in faculty examinations, to have kept active interests in the classics, in the committees of the Undergraduates' Association and the Sydney University Medical Society, in Saint Andrew's College (where he was Senior Student) and as editor of *Hermes* (the official magazine of the undergraduate body) would make one doubt that there were only twenty-four hours in the day. However, to this list he added the enviable distinctions of stroking the eight in his fresher year (perhaps a record), and in 1917 of captaining the first Rugby fifteen. He also represented his university in the rifle shooting team, and incidentally was a strong swimmer and capable horseman.

In 1913 he was informed by two of the selectors for the Rhodes scholarships that this distinction would be awarded him if he made application. The reasons which had prompted him to forgo the Cooper scholarship urged him reluctantly to surrender this opportunity. The decision was reached after the late Dr. E. A. Falkner of Toowoomba had asked advice from the great Sir William Osler, and the latter, having considered all circumstances, had advised against his applying.

In his third year of medicine, during which he held a prosector's appointment, medical students were not permitted to enlist with the Australian Imperial Force. Despite the fact that the emergencies of war resulted in a shortened, crammed final year, during which he had served full time in lieu of a resident medical officer at the Royal Alexandra Hospital for Children, Woodward proceeded to graduation very high in the honours and prize list. He then elected to take a short intensive post-graduate term at the Coast Hospital rather than the usual resident year at one of the teaching hospitals. This permitted an earlier acceptance for the Australian Imperial Force, and he was duly posted as regimental medical officer of the 26th Infantry Battalion, and later to the 8th Field Ambulance.

His active service in France, during a very "sticky" period at Bullecourt on September 29 and 30, 1918, was distinguished by the award of the Military Cross. The accompanying citation, embodied in a letter from General Sir William Birdwood, General Officer Commanding Australian and New Zealand troops, reads as follows:

> You were forced to establish your Regimental Aid Post in an open trench, which was subjected to continuous artillery and machine gun fire, and displayed untiring energy and courage, in tending the wounded without cessation during this period. Though your Regimental Aid Post received several direct hits, which caused casualties, you persevered in your work and encouraged your men with your fine example of coolness, and total disregard of your personal safety.

At the conclusion of hostilities Woodward spent some time in work and study, mostly in London hospitals, under an army scheme which gave Australian Imperial Force medical officers six months for post-graduate work before they were returned home. He fulfilled the course of study prescribed for the Fellowship of the Royal College of Surgeons, but as a further six months had to elapse before he reached the age of twenty-five years—the minimum age for the examination—funds compelled his leaving for Australia without the chance of presenting himself as a candidate. During this time he had been offered a place in the Australian Imperial Force training squads for both rowing and Rugby, but adjudging that the demands of study precluded his acceptance, he declined both.

Returning home he married Elizabeth Blanche Dines McDougall, of Toowoomba, to whom he had become engaged before going overseas, and commenced general practice at Emmaville. He then moved to Grafton, where he remained in practice till the end, first in partnership with his life-long friend Dr. Eric Holland, and later in a group practice, which was probably the first attempt in Australia at group practice as we understand it today. Subsequently, when his disability rendered general practice beyond him, he devoted his last years to pathology.

On one occasion, when someone remarked that it was strange that he had given his life to country practice, he slyly replied: "Many of the great men have preferred the provinces." On another similar occasion his rejoinder was: "I like looking after the sick."

It was during the last twenty-five years of his life that I came to know and understand this rare and lovable character. In retrospect I have felt it, as one might in wandering through a landscape of natural beauty, unaccompanied by officious guide or district-proud inhabitant. As each new vista unveils itself spontaneously, so did his wisdom, his humanity, his humour, vouchsafe itself in ever-varying vignettes. So innate and natural was his modesty that the recognition of his genius, and the liberality of his knowledge, were to me a gradually unfolding process. Truly, in him was Bacon's summing of the values of reading, writing and discourse exemplified, and the worth of the study of the humanities demonstrated.

During his years at Grafton his success was not merely that of the full consulting room. He had the esteem, affection and respect of partners and fellow practitioners in full measure. To his patients he was indeed the beloved physician, and even when his health was failing he gave of his best, regardless of their social or financial status.

In him was the perfect doctor-patient relationship ideally displayed.

The trust this engendered is shown in the following incident, related to me by one of his close colleagues.

Woodward had visited one of his less affluent elderly woman patients who was suffering a minor illness, ordered treatment, and enjoined her to stay in bed, till he called again. Taken ill himself, he returned some ten days later, to express concern that she was still abed. With the "faith that removeth mountains" she replied: "But, doctor, you told me to stay here till you came again."

Of his infant patients he was wont to remark: "I love looking after my little seedlings."

As one would expect, the delights of study remained a natural habit, and he found time not only to continue the cultivation and enlargement of his garden of classical planting and general reading, but also to further his medical standing by satisfying the examiners for his Membership of the Royal Australasian College of Physicians, and by contributing original writings in clinical pathology. It was typical of his industrious mind that his study for the M.R.A.C.P. should have been performed during a period when he had been ordered complete abstention from practice.

In his busy life the multiplicity of interests he contrived to serve is rather amazing. He was a past president of the North Eastern Medical Association and a committeeman for several terms. To his home city he devoted time on committees of the Grafton Library and the local Fitness Council. He had also been a prime mover in the formation of the "Odd Volume Club", an organization devoted to discussion on books. The minutes of this society were kept in verse and make good reading. His humanism was expressed as a founder and practical supporter of "Rathglen" Children's Home, as a Rotarian and as a member of the masonic fraternity.

Social and sporting interests demanded their quota of his attention. As president for many years of the Grafton Club, and the Clarence River Jockey Club, he was a dignified and charming host. As a racing owner he was actively aided and abetted by his close friend, Dr. Holland, by his brother-in-law, Mr. D. W. McDougall and, I fear, by his wife, whose family knew and bred good horses. This group upheld that highest sporting reputation so desirable in racing circles, but alas! not invariably found. They had some notable successes, as well as the reverses usually referred to *sotto voce*.

While Woodward was president of the Northern Rivers Racing Association for some years, I had ample opportunity, as a committee member, of appreciating his firm, wise and friendly conduct of meetings, which at times required tact and patience. A favourite expression of his in tense situations was: "Have a row if you like, but don't have a feud." I doubt if the sun ever went down upon his wrath.

While health permitted he made his solid knowledge of rowing available as coach to the Grafton Rowing Club, and we can be sure that his sunny personality left its benign influence on many of those young men. In his leisure moments one could affectionately class him as a "good mixer". He had also played golf with the Grafton Club.

Always a lover of the land, he had purchased a farm at the junction of the Clarence and Orara rivers, with the object, as one might expect, of developing it by pasture improvement. However, he retained a lasting affection for the district of his boyhood, and till the last still loved the west.

He has left behind much beautiful verse, and it is to be hoped that this legacy to our literature will be preserved in published form. Preliminary steps to this end are being undertaken.

About 1929 my contact with him was very frequent, as we were both serving with the 15th Light Horse Regiment. I really commenced to learn what a wealth of character, culture and wit lay beneath his pleasantly placid surface. He later commanded the unit, and as his second in command I realized that he was giving to military business the same care as to any other subject he set out to learn. During his term as commanding officer the regiment maintained a high degree of efficiency, winning the Forster Cup for the best machine-gun troop with record shooting each year. There were no incompatibilities in his judicious dispensing of discipline and camaraderie. He was later awarded the Efficiency Decoration.

When the Second World War erupted he volunteered for service in the Australian Imperial Force. Greatly disappointed on being rejected medically, he remarked: "Why did they ever invent a sphygmomanometer?" He was later accepted for the Volunteer Defence Corps, becoming a battalion commander and later brigade commander.

During his closing years he was relentlessly trailed by progressive ill health. To one accustomed to moving round in professional and social activities, this must have proved an almost insufferable ordeal. Never at any time did I know him to complain or lose that patience and bright wit which were such an integral part of him. His married life had been exceptionally happy, and the constant care and devotion of his wife in the many serious exacerbations of his illness did much to grant him a few extra years. Interest was also centred in the budding careers of his family, and he was spared to see these in the flowering. His son Bill, after serious bouts of illness sustained during service with the Australian Imperial Force, graduated in medicine at the University of Sydney, securing top place and second class honours in the final examinations and also the coveted Rhodes Scholarship. His daughter Barbara completed her arts course with an honours degree, eventually becoming a distinguished play and radio script writer.

Woodward's first really serious cardiac attack smote suddenly one night during a sojourn in Sydney. Fully aware of its portent, he asked to be lifted up so that he might see the lights of the harbour. At the same time he quoted, whimsically, O. Henry's last words: "I'm afraid to go home in the dark."

Though he frequently referred to John Hunter's oft-quoted statement, "My life is in the hands of any rascal who chooses to make me angry", he had, nevertheless, a faculty for loading common medical statements apropos his own ailment, with a flippant affix, as if to divert one's sympathy from his suffering. In such vein he remarked: "A man is as old as his arteries—it says so on the fruit salts bottle."

It was foreign to his nature to break off the fight, as witness this extract from a letter to my wife a few short weeks before his passing: "I have given away all thoughts of an early demise though Betsy and I have just had the 'flu, a miserable pain, but now just about all over." In a serious relapse, asked by his daughter to get better, he replied: "I'll try." Later, when distressing hiccups commenced to torment him, he wryly quipped: "They 'snuck' in under the fence."

His love of the classics sustained him, even when, realizing that twilight was stealing on, he was still very conscious of life's sweet music. How beautifully he expressed this in one of his last poems:

I too have dwelt in Arcady
And heard the piper in the glade,
And still a gay flute trills for me.

The piping shrilled and fell, and we
Danced in the sunlight, boy and maid;
I too have dwelt in Arcady.

So youth then swayed in ecstasy
To lilting tunes the piper played;
And still a gay flute trills for me.

The music falls to minor key:
I wait the last note undismayed.
I too have dwelt in Arcady.

The dance is done, and time takes fee
Of youth, to write the flautist paid;
And still a gay flute trills for me.

The low sun through the olive tree
Makes of our green a chequered shade;
I too, have dwelt in Arcady
And still a gay flute trills for me.

Scientists have satisfied themselves that matter is indestructible. To me it seems illogical, nay impossible, that the inspiring animus of Ted Woodward, whilst leaving its influence a potent force among us, should have ceased to function beyond the undiscovered bourne. The talents entrusted to his keeping he duly increased, and built into a philosophy which promoted harmony among men and sustained him in his tribulations. They rendered him an outstanding ornament of humanity and of his profession, one who has most fully merited our sincere gratitude, which we express today in this commemoration of his life and work.

GASTRIC ULCER—SOME INTERESTING ASPECTS.[1]

By T. M. Greenaway,
Sydney.

Peptic ulcer has been the subject of much investigation over the past fifty years, and although a great deal of knowledge has been accumulated, the actual cause of the process remains unknown. Many interesting aspects of this work have occupied the minds of distinguished investigators—for example, the mechanism of the pain, the cause of some of the acute complications, the varieties of treatment, both medical and surgical. Many of the remarks which I make today apply to peptic ulcer in general, including both lower œsophageal, gastric and duodenal. In the main, however, I wish to stress some of the problems in diagnosis that may be presented by gastric ulceration.

Students are taught that gastric ulcer is associated with pain shortly after the ingestion of food. With duodenal ulcer the pain is said to occur several hours after the taking of food, and in fact, is relieved by food. This is not always so. It is more correct, in my view, to speak of the typical history of duodenal ulcer, and to recognize the more variable story with gastric ulcer. For example, a gastric ulcer at the cardia may be associated with dysphagia and give quite a different picture from one situated near the pylorus.

The great majority of gastric ulcers are situated on the lesser curvature of the stomach, and in fact it may be said that if an ulcer is found on the greater curvature malignant change should be suspected. Typical peptic ulceration is seen occasionally in the œsophagus in cases in which so-called ectopic gastric mucosa exists. These lesions are rare; but ulceration, or at least inflammation, of the lower end of the œsophagus may occur in the condition known as reflux œsophagitis. The recognition of the importance of lesions at the cardio-œsophageal junction has led to a great deal of attention being paid to this region by radiologists. Few if any radiologists now would radiologically examine a stomach with a barium meal without examining the patient both in the prone position and with the table tilted head down. In this way the existence of hiatus hernia may be found and an explanation given of many a puzzling story. The confusion that sometimes exists in the minds of clinicians between so-called cardiac pain and the distress that is in fact due to hiatus hernia is well known. My own impression is that this confusion with cardiac pain is rather rare with hiatus hernia; but it is of the utmost importance not to label a person as a sufferer from *angina pectoris* when in fact the condition is due to such a lesion. At this stage it may be said that possibly in the great majority of cases of hiatus hernia the condition does not produce symptoms. None the less, it is important to bear this area in mind as a cause of symptoms that are sometimes mistaken for those of coronary artery disease. Needless to say, reflux œsophagitis with symptoms, and particularly ulceration, is associated with hyperchlorhydria, as are most ulcers, and the medical regime of sedation and antacid therapy in most cases gives good results.

A comparatively small ulcer penetrating fairly deeply, as ulcers often do, and situated high up on the lesser curvature, can cause acute and even alarming symptoms and at first present quite a problem in diagnosis. Let me illustrate this:

A man, aged fifty-two years, was stricken with sudden pain, as he described it, "just below the breastbone", about 4 o'clock one afternoon. He sweated and was apparently in a state of collapse, according to onlookers. He was taken home, and was still in great distress and remained so for one to two hours. When his doctor examined him, he thought he had had a coronary occlusion. His blood pressure, which had been known to be of the order of 140 to 150 millimetres of mercury, systolic, and 90 to 100 millimetres, diastolic, was recorded at the time as 100 millimetres of mercury, systolic, and 60 millimetres, diastolic. He was given an injection of morphine and had an easier though somewhat restless night. He had another attack of pain on the following day, and because of equivocal changes in his electrocardiogram was kept in bed for some weeks. Serial electrocardiograms failed to show any significant diagnostic changes, and the diagnosis was obviously open to doubt. Only then was it found that many years previously he had had some troublesome "indigestion" lasting for six to twelve months. Careful X-ray studies with a barium meal revealed a small ulcer niche only a few inches from the cardia, high up on the lesser curvature and situated somewhat posteriorly. This man was rested in bed for a few more weeks on an antacid regime with sedation, and had no return of symptoms. Further barium meal X-ray studies revealed no evidence of any ulcer, and the response to the occult blood test has been repeatedly negative. He is symptom-free after two years, but remains on antacid therapy.

It will be argued that this is not a typical history of gastric ulcer. This is admitted; but the point is that such sudden attacks due to peptic ulceration do occur and are a cause of great confusion and great anxiety to both patient and doctor. American writers refer in fact to the so-called "tabetic" type of ulcer crisis. In other words, they recognize the attack as one of sufficient severity to remind the observer of the severe abdominal pain of the tabetic crisis. One may say in passing that this manifestation of syphilis is very seldom seen these days. None the less, one records the term as a classic reference to a well known phase of the symptomatology of gastric ulceration. Another term frequently used is that of "biliary" crisis. This, of course, has nothing to do with biliary colic, but sometimes leads to the erroneous diagnosis of cholecystitis or cholelithiasis. Whilst a so-called biliary crisis may be commoner with duodenal ulcer, one has seen several cases in which the initial pain of gastric ulcer was so severe and the rigidity so persistent that acute cholecystitis was diagnosed. It is true that in three such cases the ulcer crater was lower down on the lesser curvature, but still at such a distance from the anatomical site of the gall-bladder as to make the confusion, when viewed in retrospect, rather remarkable. One wonders how often a gall-bladder is removed unnecessarily, and the patient treated at a later date for gastric ulceration. All too often do we see a gall-bladder removed because of a so-called abnormal response to Graham's test, and the patient still suffering from recurrent dyspeptic and epigastric distress. Of course there are many reasons for this apart from my theme of missed peptic or gastric ulceration. However, in passing one may say that we shall all be well advised seldom to urge cholecystectomy even with the so-called "pathological Graham's test" unless there is evidence of biliary colic, acute inflammation of the gall-bladder, or biliary obstruction. Just what determines these acute attacks of severe pain in gastric ulcer, often lasting several days, is a matter of conjecture. It is possible that some ulcers are associated with a greater degree of gastritis and possibly some degree of perigastritis. It is also possible that at the base of the ulcer vascular occlusion takes place, and this in itself can be, as with coronary artery occlusion, a cause of very severe pain.

More puzzling still are those patients who complain of episodes of severe pain, sometimes lasting a day or two and sometimes only a few hours, the pain being referred posteriorly, commonly to a site just beneath the left

[1] Read at the annual post-graduate course of FOCLA, Lismore, August 13 to 17, 1956.

scapula and occasionally to the left shoulder; in the case of at least two patients whom we have studied it was referred to the left clavicle and up the left side of the neck. One can only surmise that some referred pain mechanism, probably due to irritation of structures closely adjacent to the diaphragm, is responsible for this.

In one patient under our care the existence of some degree of hypertension together with aortic reflux led to the consideration for a while of a diagnosis of dissecting aneurysm of the aorta. It was not until the barium meal X-ray examination was repeated by an expert radiologist that a small but deeply penetrating ulcer was seen high up on the lesser curvature posteriorly. This was confirmed gastroscopically, and healed as rapidly as most of these ulcers do when the patient was put to bed and given appropriate antacid therapy. After a story lasting nearly a year of repeated and very severe attacks of pain, she recovered completely within six weeks, and has been well for the past year.

I have rather stressed the unusual presentation of ulcers appearing at the posterior and upper aspect of the lesser curvature; but of late attention has been given to another site of peptic ulceration which causes great confusion. This is the so-called pyloric channel ulcer. The ulcer itself is often extremely small, and it takes a wide-awake radiologist to discover it. The symptomatology here is anything but typically "gastric". It is true that most cases present with an initial period of nausea, but then the stimulus as it were seems to travel caudally, and a severe episode of small bowel colic may result, giving rise to much confusion in diagnosis. The importance of an accurate diagnosis, of course, is that these small ulcers, like ulcers elsewhere, will respond quickly and completely to the appropriate treatment—that is, rest in bed and antacid therapy.

One of the best examples of a condition causing great diagnostic confusion is the splenic flexure syndrome. This has obviously nothing to do with my subject of gastric ulceration, but I mention it because the patient often complains of apparent gastric distress, and the reference of pain is in many cases constant, giving rise at times to confusion with cardiac distress. Moreover, it may mimic some of the pictures I have tried to outline which are in fact caused by ulceration of the upper third of the stomach. The mechanism here is a collection of gas, caught, as it were, in a sort of inverse "plumber's trap" presented by an acute angle at the splenic flexure. It is commonly seen in people with a redundant ascending portion of the transverse colon. A large amount of gas may distend this area causing pressure on neighbouring structures, mainly the stomach and the diaphragm. Pain is referred towards the precordium, to the lateral aspect of the thorax, and even as far as the left shoulder and left arm. Patients often complain much more bitterly about their "indigestion", and emphasize it by the fact that they can get relief by belching. It is more diagnostic, and usually to be ascertained on careful questioning, that they get equal relief—in fact, far greater relief—from the tension after the passage of large amounts of flatus.

Thus it will be seen that what is in fact an eminently curable condition—that is, a relatively acute gastric ulcer—may sometimes be mistaken for serious maladies such as angina of effort, coronary occlusion, and even thoracic disease. It goes without saying that malignant disease must always be excluded, though severe pain is uncommon in gastric malignant disease until quite a late stage. It is always justifiable to wait at least one to three weeks and observe the rate of healing by careful X-ray studies before deciding upon operation. It is uncommon for occult blood to persist in the fæces after the first week in the case of a healing ulcer. It will be commonly present when the ulcer is malignant. However, even the undermined edges of a large ulcer, thought to be malignant, may present quite a different and more reassuring picture after a few weeks' rest in bed.

In my view the treatment in most of these cases is simple—simple, that is, if certain conditions can be adhered to. The most important of these is complete bed rest for at least three weeks and sometimes six weeks. Surgeons have often remarked upon the almost complete disappearance of the ulcer at operation—recurrent and a source of trouble for many years—which had been under treatment, for example, with a "milk drip" for but ten to fourteen days before operation. I have no doubt about the place of operation in the treatment of such a chronic ulcer. Nor is there any doubt about the place of operation in the treatment of an ulcer which has caused pronounced cicatricial narrowing of the stomach, hour-glass contraction *et cetera*, or in which there is any suspicion whatever of malignant disease. None the less, the fact remains that most gastric ulcers will heal if appropriate treatment is conscientiously carried out. I could never subscribe to the view put forward by a distinguished American visitor of only a few years ago who stated that "surgery would only be occasionally advisable in duodenal ulceration—but always to be advised in gastric ulceration". This, I assure you, is not an exaggeration of his statement. I believe I am right in saying that most of us believe the reverse to be true.

The fundamental importance of rest seems obvious—and yet it is so obvious that it is sometimes overlooked. Hilton in 1863, in his classic book "Rest and Pain", emphasized the greatly increased rate of tissue repair when complete rest was afforded. Perhaps nowhere else in the body is this seen so dramatically even within a week or two as in the case of gastric ulcer. One can literally see the ulcer healing in repeated X-ray films, and if necessary the healing can be confirmed gastroscopically. It must not be thought that the disappearance of the crater indicates healing, but it is a very good indication, and the patient can be allowed greater freedom when this happy state of affairs occurs. A few further weeks, however, of comparative rest must be allowed even after X-ray evidence of healing is obtained. A lot has been written about the "ulcer temperament". Suffice it to say that most of these people are tired, and some of them are temperamental, and a number of them have been under emotional stress. I think it is of the utmost importance to put them to bed and give them a sedative such as phenobarbital, one-half grain three times a day. In other words, they should be treated with the same degree of sedation as we would treat, for example, a thyroid patient awaiting operation. I believe that the first week or ten days in bed is of the utmost importance, and that complete physical and mental rest should be obtained. As for diet, I am delighted to see the swing so completely away from the old-fashioned ideas of ulcer diet. I agree most emphatically with a recent contributor to a British journal who wrote, after a consideration of more than 100 cases, that "in few cases if any should the ordinary hospital diet be qualitatively or quantitatively altered". In other words, let the patients eat what they will, but remember that it is wise to introduce small feedings in between the ordinary meals. This in itself has the effect of making them eat a little less perhaps with their main meals. As for antacid therapy, I believe that they should become "Amphojel"-minded. It probably does not matter what particular antacid you use, but I would warn you against relying on the tablet preparations. A number of my intelligent patients have followed my direction and have watched their stools. You would be surprised to know the number of such tablets that are completely untouched by the digestive process. It is obvious that if the patient has been in much pain, and particularly if the ulcer has been associated with a good deal of surrounding gastritis, his appetite will not be restored for some days. These are the patients for whom common sense possibly demands the use of a gentler introduction to the full diet. However, I cannot deplore too much the idea that a gastric ulcer patient must perforce have an "ulcer diet" and in fact remain on a so-called ulcer diet all his life. I believe nothing to be further from the truth. It is much more important to keep them taking some gentle sedation for many months afterwards, and keep them taking "Amphojel" possibly for many years. In other words, medical science so-called, after many years of trial and error, is at last coming round to the wisdom of the man who in "Macbeth" wrote more than three hundred years ago:

> Now good digestion wait on appetite and health on both.

INDICATIONS FOR SURGERY IN PEPTIC ULCER.[1]

By Evan Thomson,
Brisbane.

I have been advertised to speak on two subjects—"Indications for Gastrectomy" and "After Gastrectomy". As brief titles commonly do, these say too much and at the same time too little. On the one hand I intend completely to ignore conditions such as tumours for which some type of gastric resection is indicated, and on the other hand I wish to deal, not only with gastrectomy, but with some other aspects of the wider field of "surgery in peptic ulcer". I intend to deal predominantly with those aspects of the subject that are of vital interest to the general practitioner. He has to deal with these patients both before and after operation. He frequently has to make the initial decision directing them to the surgeon, and he is often responsible for supervision of much of their late after-care. I shall therefore omit much of the technical side of the subject, and only touch on some principles of these aspects as they affect the more general problems before us. I intend to consider first the indications for surgery, and second the results, complications and sequelæ of surgery with particular reference to gastrectomy.

Broadly, the indications for surgery are connected with (i) acute perforation, (ii) obstruction, (iii) hæmorrhage, (iv) intractibility and (v) anastomotic ulcer. The first four will be discussed; a detailed discussion of anastomotic ulcer would be out of place here.

Acute Perforation.

Most of us were taught that acute perforation of a peptic ulcer demanded surgical intervention with all speed unless the patient was so ill that our operation would only hasten his death. However, we do well to remember that many eminent authorities rely on gastric aspiration to secure sealing of the perforation in most of their patients. A second problem concerns the operation to be performed should surgery be undertaken—should it be simple suture or gastrectomy? I do not believe that a simple answer to either of these questions can be given that will have universal application. The type of surgeon available, the efficiency of the nursing staff and the facilities at hand will profoundly affect our decision.

Hermon Taylor has been responsible for much stimulation of British interest in the conservative management of acute perforation; but I think we do well to remind ourselves that his figures were secured under ideal circumstances, and that they are in reality a tribute to the energetic devotion of his hospital staffs. When we have not available his desirable set-up, it may not be so wise to follow his advice. Circumstances will clearly dictate our policy. Were I called upon to treat a patient with an acute perforation at sea, I should be happy to follow a conservative course if the adequacy of aspiration could be assured by personal supervision. On the other hand, many of us are forced to treat such patients in the midst of an over-busy life and in hospitals where the adequacy of aspiration cannot be guaranteed, when it is of necessity entrusted to inexperienced nursing staff. Under these circumstances—and this will, I think, apply to most of us—operation is to be preferred. Operation is also of course indicated if the diagnosis is in real doubt, if there is evidence of gross peritoneal contamination, and if improvement does not follow institution of the conservative regime. Two recent cases confirm me in my view on this matter.

A patient with a known chronic duodenal ulcer, which had perforated on a previous occasion, underwent operation about 10 p.m. for a second acute perforation. The ulcer was large and the surroundings were grossly indurated. Closure of the perforation was extremely difficult, and the hole was ultimately plugged with omentum. Instructions were left that particular care was to be taken to keep the stomach empty by aspiration through a wide-bore tube. When I visited this man some twelve hours later I was astonished to find that nothing at all had been aspirated from his stomach, and while I was there he proceeded to vomit large quantities of gastric content.

The second patient underwent gastrectomy. The next morning the sister in charge reported that nothing had been aspirated from his stomach. She was most surprised when I proceeded to withdraw over 12 ounces.

Both these patients were nursed in reputable private hospitals by experienced nursing staff. In one case continuous suction was employed, and in the other intermittent aspiration by syringe; neither was efficient.

That we can with justification vary our policy in this fashion is, I think, indicated by the results of the two methods of treatment. Broadly, the overall mortality is much the same with the two methods of treatment when they are both carried out under ideal conditions. Hermon Taylor has pointed out that perforation of an acute ulcer rarely proves fatal under either policy. Deaths occur in perforation of chronic ulcers, and with either form of treatment the mortality is somewhere about 15%. Taylor has now adopted the view that perforation of acute ulcers should be managed conservatively, and that if patients with perforated chronic ulcers are operated upon, gastrectomy should be performed; but I should like to repeat that, in my judgement, to apply this policy to all Australian conditions would be to court disaster.

I should like to commend for your thoughtful consideration another significant difference in behaviour between acute and chronic ulcer. Hermon Taylor has recently pointed out that when perforations of acute ulcers are treated conservatively, subsequent ulcer symptoms are uncommon (three cases in 47), and in none of his patients has gastrectomy been needed. However, after suture at least 25% of patients develop chronic ulcer, and a good number need subsequent gastrectomy. It would appear that this difference is the direct result of suture, which both introduces a foreign body and interferes with blood supply to the ulcer edges. In other words, an acute ulcer can be converted into a chronic ulcer by suture of a perforation.

However, despite these observations I find myself compelled to advocate a different policy under most Australian conditions. In perforation of acute ulcers I believe operation should be undertaken in most circumstances. Could I sit by the patient's bed and be certain that aspiration was adequately performed, I should be happier. I am not happy at the present time about the adequacy of aspiration in either private or public hospital practice. Operation is also indicated in perforation of chronic ulcers for all patients who can stand it. Again I believe that in general simple closure is the procedure of choice. For the experienced gastric surgeon in suitable surroundings and with the right ulcer, gastrectomy undoubtedly has a place, and for technical reasons this place will be greater in gastric than in duodenal perforation.

Obstruction.

We well know that obstruction may occur in gastric ulcer leading to hour-glass contracture, and in duodenal ulcer leading to pyloric obstruction—a rather unfortunate term, in that the obstruction is rarely pyloric, but is usually in the first part of the duodenum. We also well know that œdema, spasm and scarring can all be responsible. Of these conditions I want to say very little. If operation is indicated in hour-glass contracture of the stomach, gastrectomy is the procedure of choice, the stomach being cut, of course, above the ulcer. In gastric outlet obstruction, conservative treatment may be indicated if it is judged that œdema and spasm are responsible, but the majority of such patients will ultimately require surgical aid. When operation is needed, both gastrectomy and gastroenterostomy have their place. The latter operation is indicated for females and for some of the older males. However, I always remember very vividly that the most rapid stomal ulcer I have seen developed after gastroenterostomy for obstruction in a man well in his seventies. I need scarcely remind you that the fractional test meal examination is not a reliable guide in these cases. Outlet

[1] Read at the annual post-graduate course of FOCLA, Lismore, August 13 to 17, 1956.

obstruction leads to chronic gastritis and this to a reduction in the acid level, which may rise once again when the gastric mucosa recovers after gastroenterostomy.

Hæmorrhage.

As a broad statement it is clearly correct to say that treatment of bleeding ulcers is primarily conservative, but that in selected cases operation is demanded if the patient's life is to be saved. However, I hasten to point out that such surgery is only for the experienced gastric surgeon. The "occasional gastrectomist" is calculated to bring surgery into disrepute. Circumstances must be good, resuscitation services adequate and specialist anæsthesia available. Our aim in selecting these patients for operation is to decide which of them are having hæmorrhage from vessels unlikely to seal spontaneously and permanently, and there are many factors that we have to consider.

Certainty of the Diagnosis.

We would do well to operate only on patients who have a proven ulcer or a history that leaves us in little doubt that one exists. If we operate indiscriminately on all patients without making this distinction, we shall again bring surgery into disrepute. Remember that hæmorrhage is not a contraindication to X-ray examination with the barium meal, or to gastroscopy if you like that form of examination. In some cases of unproven ulcers and of acute ulcers and erosions, operation is judged necessary at times by the persistence or recurrence of massive hæmorrhage; but in these cases we should be rather more careful, for although we undoubtedly save some lives, the functional results of our surgical procedures are frequently unsatisfactory. One of my most uncomfortable patients had gastrectomy performed for massive hæmorrhage from an acute ulcer high on the lesser curvature of his stomach which had produced no previous symptoms. It is, of course, generally true that the worse the patient's symptoms before gastrectomy, the better will he be after. Many of the subsequent factors are of help in allowing us to guess the probable nature of the bleeding point.

Age.

Death is uncommon under the age of forty years, although it does occur. In patients aged over forty-five to fifty years, larger arteriosclerotic vessels and chronic ulcers are more likely to be implicated. Operation is indicated at times at all ages, but is indicated in more patients aged over forty-five to fifty years, until, of course, it is again contraindicated by the excessive age of the patient. My youngest patient was aged eighteen years, and was suffering from hæmorrhage from the splenic artery, which had been eroded by a massive gastric ulcer.

Site of the Ulcer.

I have been more ready to operate on patients with bleeding gastric ulcers than on those with duodenal ulcers. The mortality rate from hæmorrhage from gastric ulcers is greater than that from duodenal ulcers, although the difference is not great.

The Presence of Hypertension and Other Evidence of Arteriosclerosis.

It is doubtful whether evidence of generalized arteriosclerosis bears any relation to sclerosis of gastro-duodenal vessels. It probably does not, but this factor is important for other reasons. Anæmic anoxia affects cerebral, cardiac and renal tissues, and more so if their vessels are arteriosclerotic. Renal failure is an important cause of death in hæmorrhage, and such patients are more likely to develop it.

Duration of the Ulcer.

The longer the ulcer has been present, the greater is likely to be the scarring about the bleeding vessel preventing retraction of its wall.

Massiveness of the Initial Hæmorrhage.

Evidence of massive initial hæmorrhage may be present before the practitioner's eyes. Otherwise the patient's reaction alone can guide us. The hæmoglobin value is unreliable, and estimations of blood volume are not easy to perform. Blood pressure levels may be the best guide. Note at this point that hæmatemesis carries a worse prognosis than melæna alone.

Continuing Hæmorrhage.

Our assessment of the patient with continuing hæmorrhage depends on continued vomiting of fresh blood, on continued aspiration if we have a tube in place in the stomach, and on the pulse rate and blood pressure recordings.

Recurrence of Hæmorrhage.

Should hæmorrhage recur after ceasing during treatment, operation should be seriously considered. More than one recurrence of hæmorrhage in hospital in an older patient is strong indication.

History of Previous Hæmorrhage.

Particularly if previous hæmorrhage has been severe, such a history will influence us in favour of operation.

The Nature of the Symptoms Experienced Prior to Hæmorrhage.

Symptoms experienced prior to hæmorrhage may indicate a recent extension of the ulcer. An acute exacerbation of pain with posterior radiation may indicate a posterior extension of a chronic ulcer, which may have eroded a large vessel such as the gastro-duodenal, left gastric or splenic.

Other Indications for Surgery.

Other indications for operation may already exist. For example, patients sometimes have a hæmorrhage while awaiting operation for ulcer on other grounds. Under such circumstances one is much more inclined to proceed at once.

Coexisting Pyloric Obstruction.

Coexisting pyloric obstruction is a bad combination, and is usually best treated by immediate operation.

Coexisting Perforation.

Hæmorrhage may occur just before, at the same time as, and after perforation and in each case operation is usually indicated.

Comment.

In the surgery of hæmorrhage, the timing of operation is all-important. Death of the patient in these cases frequently occurs in anuric uræmia, and the longer renal anoxia has been present, the more likely is this to occur. If operation is to be performed, the earlier it is undertaken the better. Too often the surgeon is called too late. Every patient should be examined early by the physician and surgeon together, and placed in one of the following three groups: (i) those clearly needing operation as soon as they can be resuscitated; (ii) those who are most unlikely to need operation; and (iii) those who need careful watching and who may require operation in the next forty-eight hours. I have found that it is in this third group that indecisive procrastination has been most harmful. These patients need careful observation, and should not be left to the care of the inexperienced.

The technical details of operation I shall intentionally omit, merely mentioning the difficult problem presented by the patient in whom no bleeding point can be found. Sometimes we are taxed to the very limit.

Recently I was called in consultation to a girl, aged eighteen years, who was having most alarming hæmorrhage. She was rather too ill for a completely reliable history to be secured. However, it was known that she had been under medical care for some six months, although no barium meal X-ray examination had been performed and her complaints had been taken rather lightly. Sufficient could be elicited, however, to allow us to conclude that she almost certainly

had a posterior wall gastric ulcer, and operation was decided upon. Anæsthesia was induced and a cuffed tracheal tube passed, whereupon she had a massive hæmorrhage which liberally stained the operating table and theatre floor. At this point she owed her life to the calm self-possession of my anæsthetist, who reported that the patient was completely pulseless and that no certain signs of life could be discovered, but that, if I wished to open the abdomen, she was prepared to carry on. I rapidly entered the abdomen, to find it almost filled by a blood-distended stomach. After this had been evacuated as much as necessary, vigorous bleeding from the splenic artery was found. I compressed the bleeding point digitally for some ten or fifteen minutes while blood was poured into the patient, and her pulse gradually returned. I then ligated the splenic artery by underrunning it with a silk stitch on either side of the bleeding point. The second largest gastric ulcer I have ever seen was present; it was almost six inches in diameter. Gastrectomy was quickly performed. The patient had an uneventful recovery, and is now well and free from symptoms.

Intractibility.

I cannot define adequately in a few words just what I mean by intractibility. To define it as "failure of medical treatment" would be inadequate, and, moreover, how many interpretations of this could we have! And when to this we add "likely failure of medical treatment" we are in deeper water still. Further, there is evidence that the course of ulcer disease may not be much influenced except symptomatically by medical treatment. In the very nature of the case, then, we can lay down few hard and fast rules. Each patient presents an individual problem, and must be so considered. There are, however, a number of important considerations that will influence our judgement as we decide that an ulcer is intractible.

First I should like to ask a question—should peptic ulcer be treated medically at all? The answer lies in the natural tendency of many peptic ulcers to heal, so that a medical policy is reasonable. As Ogilvie has well put it in regard to chronic duodenal ulcer, we should adopt a very conservative policy, since the natural tendency is frequently toward ultimate healing as the patient "acquires that state of mind that he calls philosophy and his wife calls laziness". However, in gastric ulcer a good case can be made out for operation in all cases upon diagnosis, but I think that we should never lose sight of one important fact. Gastrectomy involves removal of a large part of an extremely important organ, many of whose functions cannot be taken over by other organs, and it is irreversible. Such an operation must, of necessity, have some unfortunate sequelæ, and no patient without most of his stomach can be as good as one with a normally intact organ. The patient naturally compares his state after operation with that before, so that an identical post-operative state may be regarded by two patients in completely different lights, depending on what they were like before. In general, the worse the patient was before operation, the more satisfied will he be afterwards. It is a very good thing to have symptoms to relieve. I have found that the most grateful patients are those with complete gastric outlet obstruction, even though the cynic might say that they could not be worse. The psychoneurotic patients with minimal symptoms are to be avoided.

At the outset we must assess the patient as an individual, and learn as much as we can about his mental and emotional reactions. We must try to decide what proportion of his symptoms are organic and what "functional". Those with a large "functional" element are unsuitable for operation. We should attempt to discover how much self-discipline the patient is capable of exerting. Too frequently medical treatment has failed, but only because in actual fact the patient is a failure and not the medical treatment. Such a patient has often not been able or has been unwilling to discipline himself and order his life in accordance with a reasonable medical regime. He will be equally unable to discipline himself to the few restrictions of a small stomach. In general it is wise to make a patient earn his gastrectomy. On the other hand, it is equally true to say that an ulcer is intractible when it can no longer be tolerated by the patient. But it is clear from what I have said that the threshold of tolerance of the individual patient as determined by his personality is all-important. To put this problem in another way, we must try to decide how likely a patient is to suffer from symptoms due to his small post-operative stomach; this is far from easy, and we frequently fail.

One of my most satisfied and grateful patients is the husband of a former matron of a private hospital, who did little work while his wife slaved. His story was atypical, and we judged there was a large "functional" element. But he had a small pyloric canal ulcer which had been assiduously treated without effect, and I was prevailed upon against my better judgement to operate on him, largely because a pyloric ulcer is a most unsatisfactory medical problem. Much to my surprise his whole attitude changed. He secured such relief that he made his wife sell her hospital lease and retire to the home, while he has thrown himself wholeheartedly into a business venture. He is never tired of telling how his whole attitude to living has changed as a result of relief from his symptoms.

Fortunately judgement is often easier than that.

Recently I examined a man, aged about sixty years, an insufferable egotistical extrovert with a chronic penetrating duodenal ulcer which will never be any better without gastrectomy. For years he has refused to follow advice given by a veritable legion of doctors who have been temporarily in favour. A dental surgeon has done an excellent job, but has "ruined his mouth". And I am sure I could easily cure his ulcer, but just as certainly ruin his stomach. He needs operation—but by another surgeon.

Next we must carefully study the patient's ulcer if need be for a period. Each ulcer has its own natural history. This varies from the acute, rapidly healing ulcer of temporary stress, through the cyclical type of chronic ulcer to the ulcer with relentlessly advancing symptoms; and as we study the ulcer we gain some idea of what the future is likely to hold. Our views on prognosis will influence our judgement on treatment, and they depend upon our knowledge of the natural history of the disease. At operation many ulcers, particularly gastric ulcers, are healed or healing, and this is as it should be. The natural history of peptic ulcer is characterized by periodic remissions and relapses, and in many cases the major aim of our surgical procedures is prevention of relapse rather than cure of the presenting episode when we judge that relapses are most likely to be frequent or severe. In the study of the individual ulcer we shall note particularly the length of history and the frequency, duration and severity of relapses.

It is usual to be reminded that failure of medical treatment means failure of adequate medical treatment, in which all environmental, psychological, dietary and pharmacological weapons have been used. Here we have a multitude of opinions on the value of various weapons. Despite what has been written on the subject, I believe that bed rest is of value; in fact, response to treatment in bed is almost diagnostic.

Nevertheless, we must realize clearly that failure to respond to medical treatment may mean one of two things. On the one hand it may mean that operation is necessary; but on the other it may mean that our diagnosis is wrong and requires review.

How do we assess "failure to respond"? Here, again, remember that there are observer differences. We must be guided by (i) the severity and persistence of pain, (ii) the degree of loss of sleep, (iii) the time lost from work and the effect on the patient's earning capacity and his ability to support a family (we will operate on a worker earlier than on a millionaire), and (iv) the X-ray findings. This final point is important in the management of gastric ulcer, in which failure to heal is a significant finding. Let me say here a word about the pitfalls of X-ray films. "False positive" results occur, and ulcers are not uncommonly missed. I have seen a gastric ulcer six inches in diameter and a duodenal ulcer two and a half inches in diameter not shown by barium meal X-ray examination. Remember that in many cases the diagnosis of peptic ulcer is best made clinically—that is, on the history. Too often do I meet the attitude that regards a negative X-ray finding as excluding the diagnosis of ulcer.

The question of age has seen a gradual change in view over the last decade or two. Today I think that it would be fair to say that one avoids gastrectomy in patients aged under twenty years unless it is imperative, that one does not hesitate in patients aged over forty years, and that one selects rather more carefully in between.

The position of the ulcer is of the greatest importance when one is dealing with gastric ulcer. Here every ulcer is a potentially ulcerating carcinoma, at least if the history is short, and he would not go far astray who regarded all gastric ulcers as malignant until proven otherwise. We should thus operate early or at once on all ulcers in the immediate prepyloric segment or not on or near the vertical part of the lesser curvature. A short history in a patient aged over fifty years makes the ulcer very suspect and a candidate for early operation. Gastric ulcer does occur with achlorhydria, but it is uncommon, and such a combination strongly indicates operation. If we choose to treat a patient with gastric ulcer medically, we should follow progress closely by serial X-ray films and operate if healing is not proceeding rapidly. We all have our own time limits, from one month to several months; but clearly our limit must be elastic, depending on the individual case. We should, of course, never lose sight of the fact that relief of symptoms and gain in weight do not mean that an ulcer is not malignant. Nor does radiological improvement indicate this. The peptic ulcer portion of an ulcer-cancer may decrease, or the ulcer may fill with malignant cells. It is obvious, then, that we must manage our patients with gastric ulcer with particular care.

Contrary to what I was once taught, the size of an ulcer bears no relationship to its character, whether it is benign or malignant. In fact, my own experience is that in general the larger a gastric ulcer, the more likely is it to be benign. Size must be considered in relation to duration. A large gastric ulcer with a short history may heal as if by magic. A large ulcer with a long history is unlikely to heal and, particularly if there is evidence of fixation or penetration, it is usually best operated upon without the waste of any time for medical treatment.

A study of the symptoms and particularly the type of pain is all-important. I want particularly to stress that in peptic ulcer two distinct types of pain occur. The uncomplicated ulcer produces visceral pain, related to meals and relieved by food and alkalis, and of course presents no problem in diagnosis. However, the ulcer which has penetrated the full thickness of the gastric or duodenal wall commences to involve parietal structures and produces a parietal pain. This pain may be most bizarre in character, and I have seen it in the left iliac fossa, the left upper quadrant of the abdomen, the back, the chest and even the arms. It is postural in character, increased by activity and relieved by rest. It is no longer, or at least not to such a degree, related to meals or relieved by alkalis. It is now relieved, unlike the visceral pain, by "A.P.C.", and many of these patients have methæmoglobinæmia. In fact, I know one experienced physician who tells me that whenever a woman enters his room with blue lips, he thinks first of penetrating ulcer. Such evidence of penetration and the changing pattern from visceral to parietal pain are strong indications for surgery. One always makes the proviso that the diagnosis can be made first—and these cases frequently tax the very elect.

Well do I remember one woman, aged forty-two years, who for a year had been treated for coronary disease because of pain in the chest, back and arms made worse by exertion. On more careful questioning, it was apparent that no immediate relationship existed. Merely would she be worse after a day of great activity. The pain woke her regularly at 2 a.m., a very significant fact. Rather unusually, it was worse when she lay flat and was easier when she was upright. It was always made worse by housework, and again was worse after the evening meal. She had always obtained some relief from antacid powder, but only very recently had an epigastric element entered the story. Repeated X-ray films gave negative results, and one experienced physician had doubted that her symptoms were organic. At operation I found a duodenal ulcer over two and a half inches in diameter excavating both liver and pancreas.

Repeated hæmorrhages, particularly if the blood pressure is rising or if other indications exist, may indicate interval operation.

Repeated perforations indicate gastrectomy. Indeed, I believe that operation should be undertaken if an ulcer perforates twice. Significant symptoms after one perforation may, of course, require operation.

Multiple ulcers are not uncommon—both multiple gastric and coexisting gastric and duodenal ulcers. Such patients are frequently best treated surgically. I should remind you, of course, of the classical sequence of duodenal ulcer, pyloric obstruction, gastritis and stasis gastric ulcer.

SOME HAZARDS OF MULTIPARITY.[1]

By G. SHEDDEN ADAM,
Brisbane.

IN recent years, and in spite of the tendency to limit the size of families, increasing recognition has been taken of high multiparity as a clinical entity in its own right. Such patients are, of course, more commonly encountered in public hospital practice, but not exclusively so. They may be subject to a series of complications, all the more dangerous because they are often unsuspected. It is therefore unwise to act on the assumption that a long series of previous normal deliveries is an automatic guarantee that this state of affairs will continue.

As Donald (1955) has pointed out in a useful review of this subject, many factors operate to increase the hazards of high parity. For one thing the patient is growing older, and her cardio-vascular system is consequently less resilient, so that hypertensive disease is more manifest. Other general conditions which are part of the normal process of aging are likely to complicate the clinical picture. Obesity, which is often gross in these cases, increases the dangers of child-bearing, as is well known, and not the least reason for this is the difficulty of making an accurate examination.

Duncan *et alii* (1952) have over recent years given convincing evidence that sociological factors play a very important part in the hazards of high multiparity, and I do not suggest that the complications to which I shall refer are an inevitable accompaniment of high parity *per se*. We have all attended patients having their sixth, seventh or eighth child and enjoying withal a high standard of health and freedom from complications, as well as a deep measure of contentment. Nor is the mental and physical good health of such patients necessarily a measure of their economic status.

However, the other side of the picture must be faced, for many patients of high parity are poor, overworked and chronically tired. Many of them have never been free from some degree of anæmia, which may be carried over from one pregnancy to the next without respite. The care and feeding of their children are often carried out at the expense of their own nutrition, for these mothers are often "too tired to eat". They are consequently often very short of vitamins and first-class protein and tend to an over-indulgence in carbohydrate foods. They are often too busy to attend to their own health, and a succession of pregnancies and periods of lactation leads to loss of dental fitness, which in turn aggravates dietary deficiencies. Their working day is long and rest periods are often inadequate.

With increasing weight and lumbar lordosis, the abdominal wall loses what tone it may have had, and varying degrees of pendulous abdomen complete the clinical picture.

Complications of Pregnancy.

A complication of multiparity is that the abortion rate is increased, but it is difficult to say how much of this is spontaneous. Anæmia is usually of the iron-deficiency type,

[1] Read at the annual post-graduate course of FOCLA, Lismore, August 13 to 17, 1956.

and every effort should be made to see that the patient's hæmoglobin value is at least 80% before she approaches term. The method of achieving this will depend upon the time at one's disposal, and because there is often some doubt as to whether iron tablets prescribed are really taken, it may be preferable to use the intramuscular route or occasionally to make use of transfusion of packed red cells. The woman who faces labour with a hæmoglobin value of 65% or less is completely unprepared for any complications.

Hyperemesis is relatively uncommon in multiparous patients, but is far more significant and should always be taken seriously, for such a label may mask a true diagnosis of primary hepatic disorder, intestinal obstruction or hiatus hernia.

Hypertensive vascular disease comes increasingly into the picture with the patients' higher age group, and if it is present it necessitates prolonged periods of bed rest during pregnancy. Termination of pregnancy in the worst cases is sometimes advised, although I am of the opinion that with proper hospital supervision this should seldom be necessary. Nevertheless, the fœtal prognosis is adversely affected; premature induction of labour is often necessary, and these cases should not be allowed to become post-mature. In spite of the increased incidence of hypertensive disease, preeclamptic toxæmia and eclampsia are no more common in women of high multiparity; but the effects of eclampsia are more serious, and the mortality is raised because the patient is less well equipped than younger women to contend with them.

To the patient with a cardiac disability, high degrees of multiparity constitute an additional risk. The patient, of course, is older, and therefore cardiac failure is more likely. For this reason the tendency nowadays is to advise patients with cardiac lesions to have their family whilst they are still young.

As might be expected, any of the minor ailments of pregnancy are exaggerated, particularly such conditions as hæmorrhoids and varicose veins, which may be very troublesome indeed.

Twin pregnancy is about three times as common in high as in low multiparity, and this fact can be partly explained by the patient's high degree of fertility. *Placenta prævia* likewise is more common in direct proportion to the patient's parity, and because the patient may disregard small warning hæmorrhages, bleeding from *placenta prævia* may be very sudden and profuse, so that its effects, in the likely presence of iron-deficiency anæmia, are magnified. Accidental ante-partum hæmorrhage is also largely a complication of multiparity, and its consequences, both immediate and remote, contribute materially to current maternal mortality.

There is no unequivocal evidence that fœtal abnormality is more common in women of high than of low parity, although mongolism is said to be more frequent under these conditions. Sensitization of Rh-negative women through iso-immunization to the Rh antigen, with the birth of erythroblastotic infants, has greater opportunities to develop.

The high incidence of premature labour in women of high parity is partly due to the fact that multiple pregnancy is more common, but in the main it is the result of associated sociological factors.

Complications in Labour.

It is when we come to labour that the woman of high parity deserves extra vigilance. The principal potential complications include malpresentations with or without prolapsed cord, disproportion whether at the brim or in the pelvic cavity, post-partum hæmorrhage, silent rupture of the uterus, obstetric shock, precipitate labour and amniotic fluid infusion. These observations deserve more detailed comment.

Malpresentations and Prolapsed Cord.

Malpresentation, as has already been stated, is favoured by a pendulous abdomen and lack of tonicity of the uterus, together with an acute inclination of the pelvic brim resulting from associated lordosis of the spine. In spite of a normal presentation, the fœtal head may remain free after the onset of labour, and thus may be a predisposing factor to prolapse of the cord as the membranes rupture.

In many of these cases the position of the fœtus is unstable, and treatment by external version and the application of an abdominal binder is frequently ineffective. An oblique position of the fœtus with the head in one or other iliac fossa may thus persist even after labour has commenced. These cases call for the closest supervision until the head has firmly engaged in the brim. The alternative of external podalic version and the bringing down of one leg as soon as the cervix will admit two or three fingers carries with it a slightly greater risk of prolapsed cord, but may be preferred. Internal podalic version at a later stage in the labour should be reserved for small infants, such as a second twin with transverse presentation or a premature infant with a prolapsed arm. With large infants (common in women of high parity), the risk of producing a tear in the lower uterine segment by internal version is not necessarily abolished by gentleness and experience. However, it may occasionally be the only thing to do.

Should version to a breech presentation be carried out before full dilatation, as part of the treatment of persistent malpresentation, the question of whether to bring down one leg or two is debated. In our experience, if both legs are brought down the trunk often descends quite soon afterwards, and the incompletely dilated cervix may close down on the infant's neck, leading to difficulty in delivering the after-coming head.

On the other hand, when only one leg is brought down through a patulous and only slightly dilated cervix, it is not unknown for the patient to "go out of labour" or to exhibit hypotonic inertia, which occasions anxiety for the welfare of the infant. However, slight steady traction usually acts as a stimulus to effective contractions.

Stallworthy has called attention to the frequent association of recurring malpresentation with *placenta prævia* in parous women, and has been able in a considerable series of cases to diagnose the latter condition by radiological means before the first hæmorrhage.

Disproportion.

The relative frequency of cephalo-pelvic disproportion in women of high parity may at first seem surprising, as one is tempted to regard the pelvis of a highly parous patient as being of proven capacity unless all previous infants have been of less than average size. Nothing could be more dangerous than such an attitude. For one thing, there is a tendency for the mother to have larger infants with successive pregnancies, and this may consequently give rise to cephalo-pelvic disproportion for the first time. Secondly, in high multiparity two things may influence pelvic capacity unfavourably; first, the increasing inclination of the pelvic brim already referred to, and secondly the occasional forward subluxation of the sacrum upon the sacro-iliac joints, so that the sacrum advances towards the symphysis and the antero-posterior measurements, both at the brim and in the cavity, are effectively reduced. Failure to recognize this condition may end in uterine rupture, especially in the presence of unusually strong contractions. The development of varying degrees of spondylolisthesis will also affect the capacity of the pelvic brim, and I have followed through a small number of such patients. In each case there was delayed descent of the fœtal head through the pelvic brim, but in no instance was Cæsarean section required.

To meet a situation such as might develop in a parous woman with recently lessened pelvic capicity, and in whom high mid-pelvic arrest or at least delay has occurred, the concept of "trial of forceps" has recently been elaborated. Briefly, it is suggested that the term "failed forceps" need not be derogatory if the original intention of the operator was to test by this means the pelvic capacity for vaginal delivery without using undue force. If it was the opinion of an experienced obstetrician after tentative traction that such force would be required and that some degree of

trauma would be inevitable, Cæsarean section could still be safely carried out. Obviously the concept in practice would call for a high degree of judgement and could easily be abused. Wisely used, it could change the picture of a traumatizing delivery with the possibiity of a stillborn infant and talk of "a Cæsarean next time", to an early but justified decision for a "Cæsarean this time", a live infant and an undamaged mother. In other words, it could carry the principles of trial of labour for brim disproportion into the field of high mid-pelvic disproportion, with all the safeguards against unnecessary operation that apply with a well-conducted trial of labour.

Ruptured Uterus and Obstetric Shock.

Uterine rupture constitutes one of the gravest risks of high parity, and it is important to realize that it may occur after only a short period of labour. Uterine contractions tend to be better coordinated and more forceful in multiparous labour, whereas the strength of the myometrium to resist rupture is by no means increased by successive pregnancies. Moreover, the presence of an infant of larger size than previously increases the strain, and the injudicious use of posterior pituitary extract for medicinal induction of labour or after artificial rupture of the membranes is more than usually dangerous in this respect. The onset of uterine rupture does not always follow the classical text-book descriptions, and some cases are probably unrecognized and may be recorded as cases of post-partum hæmorrhage or so-called "obstetric shock".

The diagnosis is, of course, more evident in cases of complete rupture, with the cessation of true uterine contractions, generalized abdominal pain (though this may not occur until the bulging membranes rupture into the peritoneal cavity), the displacement of the presenting part from the pelvic brim, the presence of a separate tumour formed by the empty retracted uterus, and signs of shifting dullness in the flanks. With extraperitoneal or incomplete rupture involving the lower segment, many of these signs are absent; there may be a latent period of an hour or more before the clinical evidence of otherwise unexplained shock and the finding of a broad ligament hæmatoma suggest the diagnosis.

Sometimes the diagnosis of uterine rupture will be made only after the completion of the third stage of labour. Such cases will, in most instances, consist of an extension of a cervical tear into the lower segment—a previous cervical laceration, a big baby and a precipitate labour may be responsible for this.

Treatment of ruptured uterus is by laparotomy in all cases of complete rupture, followed either by hysterectomy or by repair of the rent with or without sterilization. Some types of incomplete rupture can be treated conservatively with gauze packing through the tear into the parametrium, administration of morphine, antibiotic "cover", and very slow withdrawal after forty-eight hours.

Post-Partum Hæmorrhage.

Post-partum hæmorrhage is another risk; it is often attributed to preexisting poor muscle tone in the uterus, and no doubt this factor does operate. However, this is a matter of some interest, because hæmorrhage is just as likely to occur after a labour in which the uterus has been contracting well throughout, and in fact hæmorrhage may follow precipitate labour. It is suggested by Donald that the explanation lies in the fact that only retraction will protect a woman against post-partum hæmorrhage, and that when labour has been rapid, vigorous uterine contractions may cause early partial separation of the placenta at the time of the birth, but the whole process has occurred too fast for retraction to catch up, so that bleeding may occur very early in the third stage. Furthermore, full retraction may be hampered by the increased quantities of elastic tissue in the myometrium, a condition characteristic of high parity and previous subinvolution. Adherent placenta is also more common in the *multipara*.

In other instances post-partum hæmorrhage in women of high parity may be simply a further manifestation of generally poor muscle tone, and the prophylactic use of ergometrine under these circumstances is wise. The particular importance of post-partum hæmorrhage in women of high multiparity lies in their inability to withstand hæmorrhagic shock, and life may go out like a candle with relatively little warning. It is well, therefore, that the third stage should not be lightly regarded. Moreover, unexplained bleeding or shock should always arouse a suspicion of spontaneous uterine rupture.

Precipitate Labour.

Enough has been said of the risks to the mother of precipitate labour in the multiparous woman.

The fœtal mortality and morbidity from precipitate labour, however, are another problem, and fœtal wastage and disability from this cause are thought to be greater than from protracted labour. Forceful and frequent uterine contractions can cause intrauterine fœtal asphyxia through interference with the placental circulation, whilst rapid descent of the head without moulding, particularly in the presence of asphyxia, is likely to result in intracranial hæmorrhage.

Amniotic Fluid Infusion.

A rare though serious hazard associated usually with multiparity is that of amniotic fluid infusion, until recently called "amniotic embolism". This is a relatively newly described obstetrical syndrome, and occurs typically in a parous woman in unusually strong labour, often with a large infant.

It has been suggested that the amniotic fluid gains access to the maternal blood-stream by a tear in the membranes near the edge of the placenta, or through the endocervical veins, which are opened up by even minor lacerations of the cervix during the stage of dilatation.

Clinically, profound shock occurs suddenly and unexpectedly towards the end of the labour, and is followed by dyspnœa, severe cyanosis, vomiting and restlessness. Within a few moments there are signs of acute pulmonary œdema. In fatal cases there is autopsy evidence of particulate matter, such as meconium and globules of *vernix caseosa*, in the pulmonary arterioles. However, it is now believed that the subsequent signs of this condition in those patients who recover are not due to the embolic phenomena, but rather to a thromboplastic component in amniotic fluid causing massive intravascular thrombosis with consequent fibrinogen depletion, leading in turn to an incoagulable condition of the blood.

Thus, patients who recover from the initial shock and respiratory distress may die of uncontrollable post-partum hæmorrhage. The condition may be recognized by the failure of the patient's blood to clot within one hour when incubated at 37° C., and the hæmorrhagic state is effectively treated by the intravenous injection of fibrinogen (four to six grammes) should the proper indication exist.

Obstetric Shock.

There is probably no such thing as the so-called "idiopathic" obstetric shock; but shock in obstetrics can be a very real problem. Its causes may vary from a full bladder to silent rupture of the uterus, but neurogenic and hæmorrhagic components contribute equally. Because of age, possible constitutional deficiencies, the need for corrective manipulations, the risk of spontaneous trauma and the predisposition to post-partum hæmorrhage, the woman of high parity is especially at risk in this regard. Good pre-natal care, gentleness in all manipulations, minimal blood loss in the third stage and the early recognition of shock represent the means of combating this hazard of multiparity.

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PSYCHOSOMATIC DISORDERS IN CHILDREN.[1]

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THIS lecture has been included in FOCLA's annual post-graduate course to focus attention on a large group of disorders which, though vague and hardly to be classified as diseases, are extremely important in practice. We face at once a difficulty in defining the word "psychosomatic". Perhaps one could say "emotionally produced disorders of bodily function", or "disturbances of the body which have psychological origins". In one sense no illness is without its psychological component. Certainly any illness in a child is greatly affected, firstly, by his attitude to pain, reflecting upon his early training, secondly, by the optimism or anxiety displayed by those around him, and thirdly, by the pleasantness or otherwise of his surroundings. The family physician has to take these facts into consideration when deciding whether home or hospital is the better place for treatment.

Some of the conditions we are about to discuss are extremely common and of everyday occurrence in practice, but do not receive in text-books the emphasis they deserve. One finds that medical students tend to have the "organic" approach to children's illnesses, perhaps regarding children as too young for emotionally produced disorders, although recognizing that in adults such symptoms as backache and epigastric pain may have a psychological basis.

A Tentative Classification.

I shall present an attempted classification of psychosomatic disorders in childhood, about the only virtue of which is that it is original. The various conditions listed have indeed only two things in common: they all arise in part from emotional stress, and they are all sufficiently disturbing to cause parents to seek medical advice. It will be noticed that the four groups shade into one another, from disturbances almost completely voluntary at one end of the scale to those which are involuntary at the other. It is also roughly a chronological list, with the simpler conditions in early life changing to those more complex in later childhood.

I. Gratification habits—for example, thumb-sucking, head-banging, hair-pulling, masturbation.

II. Behaviour states, with an element of volition—for example, tantrums, food refusal, sleep refusal, breath-holding attacks, dirt-eating.

III. Involuntary symptom-states, with no recognizable pathological signs—for example, abdominal pain, headache, true anorexia, insomnia, fatigue, habit-spasm, vomiting, enuresis, night-terrors, diarrhœa.

IV. More elaborate symptom-complexes, with recognizable pathological signs—for example, hysteria, asthma, juvenile migraine.

Several ætiological factors are involved in the production of these disorders: (i) emotional stress, to be discussed in more detail below; (ii) inherited predisposition—often there is a family history of the same or associated disorders; (iii) behaviour training, from parental example in early life, the so-called "psychological infection"; (iv) physical stress, by way of pain, injury or debilitating illness.

It is very important not to consider children's disorders as being due always to physical causes only, or to emotional causes only; often both factors are involved. Asthma is a good example of this.

Emotional Stress.

The common types of emotional stress to which children may be subjected vary to some extent in the different age groups.

From birth to six months one finds over-stimulation, especially in first-born children. Conversely, unwanted babies may suffer from loneliness, leading to apathy and failure to thrive.

From six to eighteen months one meets food forcing, excessive parental discipline and a variety of other parental faults which can be described as "spoiling".

In the toddler stage, emotional stress may take the form of the arrival of a younger child in the family, excessive discipline, lack of parental affection, over-protection by the parents, food forcing, a remembered painful experience, and boredom.

The early school stage involves the emotional strain of separation from home, school difficulties, lurid tales from other children, and, in the youngest member of the family, lack of encouragement to grow up.

In the later school stage (seven to fourteen years), home stresses are still important. They include over-dominant parents, broken homes, parent neurosis, lack of parental affection, lack of parental imagination (in fostering interests, friends and hobbies) and a discouraging attitude to life. School stresses include bullying by teachers, overwork, bullying by other children. Adult type anxieties based on money worries and personal relationships may also occur at this age.

There is time to discuss only some of the disorders listed in the classification, but they may be taken as examples.

Breath-holding Attacks.

Breath-holding attacks are likely to be described as attacks of going blue or black in the face or as episodes of unconsciousness. The fact that the child does not breathe may have escaped notice, and a careful detailed history is necessary for diagnosis. The attacks always arise after a period of crying, which may, however, be very brief. The crying itself stems from anger, frustration, pain or sudden fright. The attack is not entirely voluntary. There is a sort of deliberate "letting go" of emotional control, after which involuntary laryngeal spasm supervenes. Epilepsy represents a diagnostic difficulty, but can be recognized by its sudden spontaneous onset. In treatment the occasions for crying should be avoided as far as possible. The attack itself should excite no parental concern—at least none visible to the child. Regular administration of phenobarbital for some weeks is usually helpful.

Adbominal Pain.

Abdominal pain always presents a difficult problem. It usually recurs at frequent intervals for months, and while it may be epigastric, it is most commonly periumbilical and colicky. It is thus likely to be confused with the pain of indigestible food or appendiceal colic, the other two common causes. One can suspect a "psychosomatic" pain if one is confronted with an emotional type of child or parents, if there is a history of stress and other stress symptoms, and perhaps by the exact circumstances of the pain in relation to the stress. A school child may possibly develop abdominal pain early in the morning, but never during the holidays or at week-ends. Organic pain is likely to be related to tangible causes, such as meals or exercise, or to appear for no particular reason, as does appendiceal colic.

One may quote the case of a boy, aged twelve years, examined in February. Since the beginning of 1955 he had been nervous and timid, suffering from abdominal pain and also from headaches and partial fainting attacks. He was a clever child, but terrified of his school-teacher, a woman, who taught him last year and was teaching him again this year, to his dismay. He had been well during the summer holidays, but his symptoms returned at once when school began again. Appendicectomy had been performed some years previously.

Anorexia.

Anorexia may be regarded as an advanced stage of food refusal. It starts insidiously, always with a "precious" child, usually an only child, often born after some years of marriage or after a long interval when the siblings are

[1] Read at the annual post-graduate course of FOCLA, Lismore, August 13 to 17, 1956.

full-grown. The parents are generally intelligent, earnest and anxious, fearful lest he will lack vitamins or other essentials. They are acutely aware of what he "should" eat. Food forcing gradually leads to increasing dislike of this foodstuff and that, finally of meals altogether and of food in general. He even cries at the sight of his mother starting to prepare a meal. The household eventually reaches the stage at which every meal is a fight, with mother coaxing, then in tears, the child struggling and screaming, food all over the place, and father muttering about home becoming a madhouse. Emotional tension runs high; and you know what happens to appetites under these conditions. The doctor may have to intervene if the patient is losing weight and to break the vicious circle of parent versus child interaction. It is sometimes essential to separate them by putting the child into hospital, though this brings its own problems of loneliness and insecurity.

For successful management a radical change in parental attitude is essential. Feeding must not be made a battle of wills, so that the child loses face if he submits and eats. On the other hand, he must not be given the impression that he is conferring a favour by eating. One aims at an unemotional presentation of food to the child in the confident expectation that he will want to copy his parents in their eating pattern. He should be allowed to feed himself, encouraged only by an occasional remark and by praise of the food by the parents to one another, not to the child. There should be no comment if he leaves some of his meal, and just minor praise if he finishes the plateful. Above all, they must lose their fear that a meal or two missed means starvation.

Habit Spasm.

A brief observation of the oft-repeated movement should serve to distinguish habit spasm from chorea. Further tests of complicated movements, such as handwriting or fastening buttons, pick out the child with chorea. One must always impress upon parents that habit spasm—and it may be a cough instead of a facial or shoulder movement—is simply the red light indicative of tension somewhere. To attack the habit spasm itself is as futile as extinguishing the red light that shines on a dashboard when the oil pressure is low.

Unsuspected tensions may have come from parents who are too ambitious—who want their son to be top of the class as well as "making" the football team—and unconsciously encourage overstrain. One meets it also in the adolescent girl who, to satisfy parental yearning for culture, learns art of speech, music, dancing and extra French in the spare time left over from school work.

Hysteria.

Hysteria usually occurs in older children and generally conforms to a fairly simple pattern, designed to get the child out of a situation that has become too difficult, and to do this without loss of face. Perhaps a case history would make this clearer.

A girl, aged eleven years, was sent to Brisbane from North Queensland about two years ago, receiving morphine in the aircraft for an intense pain in her left loin. She had suffered in this way for many weeks and was suspected of having a kidney lesion. There was, in fact, an earlier history of pyelonephritis, although on this occasion microscopic examination of the urine and pyelography had revealed no abnormality. On her arrival she could not walk or stand upright because of the pain, and lay curled up in bed. She was a nice child and evidently not malingering; but it was clear after a comprehensive examination that the pain was not of organic origin. In particular she had a curious circular patch of cutaneous hyperæsthesia in the left renal area. It then appeared that the pain had started shortly after the family had moved to a different part of the town and sent the child to a new school which she hated. Since the pain began she had been unable to attend school at all.

There are four essential stages in the management of simple hysteria in children:

1. To convince the parents that the child has no organic disease. (I am assuming that the doctor has convinced himself.) This requires careful examination and demonstration and such tests as are strictly necessary, but no more. Too many investigations give the impression that the doctor is baffled by a mysterious complaint.

2. To counteract the parents' new opinion that their child must be malingering. They are unaware of the third alternative. Stories of other hysterics help.

3. To remove the source of emotional stress.

4. To provide a face-saving way out of the trouble.

In the case just quoted, the parents, who were most understanding, accepted the first two stages quite readily. They then agreed, on their return home, to allow the girl to return to the original school. The patient brightened visibly on receipt of this news. It was then explained to her that she had a muscle spasm in the left loin causing all the pain, but that it would soon be cured by physiotherapy and infra-red light. Within a week the pain had gone and she was walking normally around the ward.

Asthma.

Asthma presents a more complex problem. Emotional stress is only one of many causal factors, along with infection, heredity, allergy, climatic conditions and others. One of the mistakes often made about asthma is to think of it in terms of single causal factors. Emotional stress in this disease operates in two ways: single episodes of stress, for example punishment at school or the excitement of a party, may precipitate an attack; or children may suffer frequently in surroundings where there is emotional strain and remain free from attacks in others. One of our patients repeatedly breaks down at home with asthma every few days, but invariably recovers in hospital and remains well as long as he is kept in. Sometimes geographical factors are given the credit for a cure, when a change in the emotional environment has really been responsible. The importance of emotional factors in asthma makes phenobarbital a valuable drug both as a component of a regular mixture and in the treatment of the attack. It follows also that we have not done our best for our asthmatic children until we have sought and corrected any psychological background stress.

Juvenile Migraine.

I am rather uneasy about presenting juvenile migraine as a psychosomatic illness. Even the title "juvenile migraine" requires some explanation. It is used here for the condition characterized by episodes of nausea, vomiting, pallor, headache and listlessness, often with abdominal pain, slight fever and acetonuria, which have been variously called bilious attacks, acidosis, and cyclical vomiting. There is a strong hereditary predisposition, and almost invariably a close relative suffers from adult migraine or has had similar attacks as a child. The episodes themselves occur spontaneously, sometimes in regular cycles, or may be touched off by dietary indiscretion; it is often noticed that stress makes them more frequent, or that a bad attack follows a particular upset.

Treatment of Psychosomatic Illness.

The broad principles of treatment of psychosomatic illness have been covered already. I should like simply to restate them in this order:

1. The doctor must convince himself that the disorder has a psychogenic or emotional basis.

2. He must convince the parents of this, assuring them that there is no organic disease, but not allowing them to feel that there is "nothing wrong" or that their child is malingering.

3. The frontal attack should be made on the emotional cause of the trouble, not aimed at the symptoms.

4. For such conditions as asthma and migraine, in which there is some spontaneity about the attacks, an attitude of philosophical acceptance must be encouraged.

5. There is a place for such drugs as phenobarbital, thiamin and *d*-amphetamine sulphate.

6. In appropriate cases the child must be provided with a face-saving way out.

THE MANAGEMENT OF BREAST FEEDING AND OF SOME CHILDHOOD MALADIES IN GENERAL PRACTICE.[1]

By STUART PATTERSON,
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NOT only does the care and management of infants and children constitute a very large proportion of our work in general practice, but, probably like many of you, I find such work the most satisfying. Babies and children soon acquire confidence and a friendliness towards the family doctor that is quite touching, and their supervision and treatment are surely the basis of our work as family doctors. The observations I make are personal ones made in private practice, in the out-patient department of the Mater Misericordiæ Children's Hospital, Brisbane, and at the Jefferis Turner Maternal and Child Welfare Home, Ipswich.

Difficulties with Breast Feeding.

I should like to commence by making some comments on breast feeding. I do so because I believe that we who are general practitioners take too little interest in this subject.

I say this advisedly. Having the medical care of an institution for the treatment of infants and young children who present feeding problems, I am convinced that many of their difficulties could have been avoided had the mothers received adequate ante-natal advice, particularly in regard to the care of their breasts.

Few of us, I believe, give any advice to our pregnant patients in regard to the care and preparation of their breasts; fewer still carry out an ante-natal examination of the breasts of these women. Yet it is surprising how much good may be done by such inspection and advice.

The nipples may, of course, be hopeless as far as successful suckling at the breast is concerned, being "tied" or even grossly retracted. However, many flat or inverted nipples respond well to daily anointing with lanoline, to being drawn out and to the prescription of a nipple shield, such as the Woolwich shield. The recommendation to scrub the nipples daily with a nail brush is now out of date; but the mothers are well advised, after washing with soap and water and applying lanoline twice daily, to draw the nipples out between the finger and thumb, at the same time rolling and twisting the nipple in much the same way as it will be treated later by the suckling infant.

The mother must also be advised to secure a suitable brassière—not one which is tight or which compresses the breasts against the chest, but one which is loose-fitting and which gives "uplift" to the breast. I am certain that many cases of failure to breast feed are directly due to the use of ill-fitting brassières, both before and during pregnancy. Such brassières cause the lactiferous ducts to be compressed and kinked, and this later leads to an inadequate or slow flow of milk to the suckling infant.

During the last two or three months of her pregnancy, the mother-to-be should be told to express colostrum from the breasts once daily. This has a twofold virtue: firstly, it opens up the ducts through which the milk will shortly have to flow, and secondly, the mother learns the art of hand-expression of milk, which may be of great assistance to her later.

After the birth of the baby, supervision of the breasts is no less necessary. The two bugbears now are (*a*) cracked nipples and (*b*) overloaded breasts.

In so far as cracked nipples are concerned, if the mother has religiously carried out the ante-natal preparation of the nipples, the danger is greatly minimized. If, in addition, it is explained to her that the baby must suck from the areola and not from the nipple when feeding, if careful attention is paid to hygiene—for example, washing of the nipples before and after feeding—and if the breasts are not allowed to become engorged, then the risks of mastitis and breast abscess are less still. I do not think the advice about bunching up the areola and putting it rather than the nipple into the infant's mouth is often enough given to mothers by the nursing staff of obstetric hospitals.

Should a cracked nipple develop in spite of all these precautions, then feeding from the breast should be stopped immediately and the infant given the breast milk, which is expressed at the ordinary feeding times. One or other of the healing creams is applied to the nipple, and the infant not returned to the breast until the nipple has healed. Sometimes a mother, at this stage, finds the use of a rubber nipple shield a comfort.

If the breast is over-distended (and this, of course, often occurs concomitantly with a cracked nipple), then we may give five milligrammes of stilbœstrol every four hours for three or four doses and relieve the breast by massage and hand expression. If inflammation or lumpiness is evident, hot fomentations are also used.

Another factor which I think of great importance in the establishment of breast feeding is a tranquil mind in the mother as well as in the infant. Again and again I see worried mothers coming into our Maternal and Child Welfare Home with grossly inadequate lactation, and after a few days of a vegetable-like existence in pleasant surroundings, their milk makes a dramatic reappearance.

One mother, I well remember, had had her lactation suppressed with stilbœstrol (on the advice of a pharmaceutical chemist, I may add), and when she came into hospital only a few drops of milk could be expressed from either breast. Yet after two weeks she was producing 40 ounces of milk daily.

Herein lies one of the chief objections to an early return home of the mother from the obstetric hospital. The practice of sending women home between the fifth and eighth days after confinement is a severe handicap, in many cases, to the establishment of lactation. Once she is home, with lactation not properly established, and probably with a multitude of domestic duties and insufficient time to rest during the day, the supply of breast milk inevitably falls.

Of course, the best stimulus to lactation is that provided by the suckling of the infant—far better than any breast pump. That is why mothers, and even nurses, must be warned about feeding, or advising to feed, from one breast only at each feed. The inevitable result, of course, is incomplete emptying of a breast, with consequent decreased milk production. Unless the milk is over-abundant, the infant should always be put to each breast at every feed, and the remaining milk should be hand-expressed to ensure that none remains.

So much for these few observations on breast feeding.

Fat Intolerance in Children.

Passing to fat intolerance in children, I was interested to read a recent article entitled "On Children who are Intolerant to Fat", by Dr. Charlotte Naish, published in *Research Newsletter* (the journal of the College of General Practitioners). Since I believe this to be a syndrome that receives all too little attention in general practice, I make no apology for speaking of it here.

In my experience these children are most often amongst the five to twelve year olds. Frequently, they come from poor homes; the reason for this is probably that the frying-pan often occupies the place of honour in the kitchens of such homes. Fried fish, potato chips, hot pies, to which liberal quantities of tomato or Worcestershire sauce are added, still constitute, with bread and jam, quite a considerable proportion of the diet of children in this country. Dr. Naish makes an observation which interested me very much in view of my preceding remarks about the use of Worcestershire sauce; she has noticed that in several cases such children have a great craving for vinegar—they will steal to obtain it. She has often wondered if vinegar aided the digestion of fat. It is certainly an interesting possibility, and one on which the biochemists might enlighten us.

[1] Read at the annual post-graduate course of FOCLA, Lismore, August 13 to 17, 1956.

However, it is not always the feckless mother who is to blame. At the other extreme is the over-conscientious mother who, believing in the efficacy of milk, dairy products and cod-liver oil, forces these down the throats of her unwilling offspring, who have often a complete inability to digest them. The effect is the same as it was in the case of the frying-pan brigade.

Here I might add a comment about the "free milk in schools" scheme. I have noticed that, in southern Queensland at all events, where many of the herd are of Jersey stock, the milk has a high proportion of fat, and this is particularly evident during and after our rainy season. Added to this, many children are in the habit of "backing up" for a second or even a third bottle of milk. Such children who present with symptoms of fat intolerance are often improved dramatically when the milk is stopped temporarily, skim milk being substituted. Later on they may be allowed full cream milk again, but in smaller quantities.

It is interesting to ask the mothers of these children suspected of having fat intolerance whether as babies they had feeding difficulty. Often the mothers will recollect that, in fact, they did, and that they frequently vomited sour-smelling, partially digested milk.

What are the symptoms of these children who suffer from an inability to metabolize fats now that they are older?

First there is abdominal pain. Often it is intense, usually it is in the upper part of the abdomen, sometimes it is in the umbilical area, and not infrequently it is in the right iliac fossa. It is not surprising, therefore, that appendicectomy is often performed on these hapless youngsters. Secondly, there is often vomiting ("bilious vomiting", the mother calls it). Thirdly, there is often an accompanying rise in temperature, sometimes a moderately high one.

Questioned, the mother will often admit that the child's complexion becomes sallow during such attacks, and also that such attacks frequently coincide with periods of nervous excitement—for example, starting at a new school, school examinations, a week-end party or an afternoon at the pictures. There is also not infrequently a family history of migraine, asthma, infantile eczema, *et cetera*, and I believe allergy often plays a part in fat intolerance. Not only do these children have difficulty in absorbing and digesting fats, but they are actually allergic to them. I believe that the allergic pain that occurs in consequence is much more common than is usually recognized. Though I am no physiologist, I have always believed that some of the pain that occurs in these cases is due to spasm of the smooth gut muscle, *plus*, in many cases, urticarial œdema of the gut's mucous membrane, with consequent retention of fæces and hence symptoms of partial obstruction.

At all events, the recommendation of a diet of low fat content and some good advice to the parents, with a suitable mixture, are usually successful. The mixture is as follows: *Sodii Bicarbonatis* five grains, *Dextrosi* 10 grains, *Aquæ Chloroformi* to one drachm; one teaspoonful to be taken thrice daily after meals. Tincture of belladonna, three to five minims, may be added to the mixture to increase its efficacy, or alternatively the following mixture may be prescribed: *Tincturæ Belladonnæ* five minims, *Elixiris "Benadryl"* to one drachm.

With regard to the diet of low fat content, I am a great believer in powdered skim milk to replace ordinary cow's milk in the diet of these children. Powdered skim milk is, in my opinion, a most useful article of diet in pædiatrics. Besides its use in fat intolerance, it is of great value in the diet of children suffering from the less severe forms of gastro-enteritis and during the initial stages of a febrile illness when full-cream milk is badly tolerated. Powdered skim milk may be given in the proportion of one part of skim milk to four parts of water, in the case of older children. Younger children may be given it in the proportion of one to five or one to six.

In the case of infants, it is well to remember the value of lactic acid milk or lactone syrup added to the milk. Their substitution for modified milk or "Lactogen" in the feeding of bottle-fed babies with this complaint is often dramatic.

Asthma.

I should like now to speak of a method which I have found of great help in the treatment of asthma in children. You will recall the division of asthma into (*a*) the allergic and (*b*) the bronchitic variety. You will recall, too, that the second of these (the bronchitic variety) differs from the allergic type in that it occurs in lungs which have previously been damaged—for example, by whooping-cough or bronchopneumonia. In the bronchitic type of asthma, besides the spasm of bronchial muscle and œdema of bronchial mucous membrane, there is much viscid secretion which has collected at the lower end of the respiratory tract.

To help get rid of this secretion, breathing exercises are of considerable benefit, and when these children are old enough to cooperate, I always send them to a physiotherapist for instruction in expiratory exercises.

But from the younger children it is not easy to obtain prolonged cooperation, and for these children I have found the use of the "breathing bottles" very helpful. These "breathing bottles" consist of nothing more elaborate than two used "Soluvac" bottles connected by rubber rubing, to the ends of which long glass rods pass down to within a half to one inch of the bottom of the bottles. Two other rubber tubes are connected to short pieces of glass tubing which extend only about one inch below the corks of the bottles. One of the bottles is three-quarters filled with water, and, by blowing into the short tube, the child blows the water into the second bottle. Then, by applying the mouth to the other short tube, the child blows the water back again into the first bottle. The child is encouraged to time the operation of blowing water from one bottle to the other, and I find the competitive element so introduced greatly encourages the child to do the exercises twice daily.

(These "Soluvac" bottles, incidentally, like ether bottles, are not salvaged for further use for the purpose for which they were designed, and they are, I find, admirably suited to the purpose I have described.)

Impetigo.

Now let me make an observation on the treatment of impetigo. Impetigo is probably the commonest skin infection of childhood.

Some years ago, dissatisfied with *Unguentum Hydrargyri Ammoniati Dilutum* because in summer in Queensland, at least in the hot weather, it was so messy, and finding that gentian violet in aqueous or spirit solution was prone to form a scab over pus and thereby perpetuate the lesion, I asked my pharmacist to try the effect of mixing *Unguentum Hydrargyri Ammoniati Dilutum* with gentian violet and putting them up in a water-miscible base such as "Dermabase" or *Cremor Cereæ Sulphonatæ*.

Such a mixture in an ointment is an improvement on either of the ingredients used singly, and the "Dermabase" softens scabs and makes the ointment easy to wash off. The only disadvantage is the colour; but this comes out of clothing and bedding with washing and exposure to sunlight. Here is the formula I use: gentian violet 1%, ammoniated mercury 2·5%, "Dermabase" to 100%. It is particularly valuable after the crusted lesions have been removed with a starch poultice.

Creams and ointments of the sulphonamides and the various antibiotics are, I believe, grossly over-used by us in our treatment of skin conditions. An ointment such as I have mentioned is far preferable for the ordinary case of impetigo.

Enuresis.

Lastly, I shall say a word or two about enuresis. Enuresis is, I believe, one of the least satisfactorily treated conditions of childhood.

By the time these children are brought to the doctor, the condition has usually been present for a number of years, and by that time the child is acutely aware of his failing and very self-conscious. This condition is heightened by the fact that the great majority of these children suffer also from a lack of security or a sense of inferiority, whether in the home or at school.

For that reason I believe reassurance of the child (and the parent) should be the first aim of the doctor. Whether it is due to local irritation (threadworms, constipation, balanitis, phimosis, acid urine or calculi), or organic causes (diabetes, nephritis, pyelitis, epilepsy, *spina bifida*), or due to allergy, bad training or mental deficiency, or to such miscellaneous factors as septic throat or teeth *et cetera*, it will, of course, be treated when possible.

But the medicine I have found most helpful was one which I started using (quite empirically) some six or seven years ago. This is it: *Tincturis Belladonnæ* five minims, *Elixiris "Benadryl"* to one drachm. If the urine is found to be highly acid, potassium citrate, 10 to 15 grains, is added. One drachm of this mixture is given in the morning and again after the child returns from school in the afternoon, and a double dose is given at bed-time. (The double dose at night avoids producing disability with vision, as it often would if taken during the day.) After a week or two the amount of belladonna may if necessary be increased to seven or 10 minims. Invariably the mother comes back with a report that in a few days there has been improvement, and after a month the wetting usually occurs only about one night in three or even less. After treatment has been continued for another month or so, the habit is usually completely broken, with only an occasional lapse—for example, in cold or wet weather and during periods of stress or excitement (examinations, parties, holidays *et cetera*).

I should be interested to know why this mixture works so well, and whether those of you who are sufficiently interested to try it find it equally efficacious.

JAUNDICE IN CHILDHOOD.[1]

By Felix Arden,
Brisbane.

With such a large subject as jaundice in childhood one cannot do much more than list the various conditions that may present with, or be complicated by, jaundice and offer brief comments upon each. As the causal factors differ somewhat with the age of the child, the following double classification may be helpful.

Jaundice in Childhood: A Classification.

(*a*) With no other symptoms:

In infancy: physiological; associated with prematurity.

In later childhood: —.

(*b*) With anæmia:

In infancy: erythroblastosis; hæmolytic anæmia.

In later childhood: acute hæmolytic anæmia (Lederer's); chronic hæmolytic anæmia (acholuric jaundice); incompatible blood transfusion; blackwater fever.

(*c*) With biliary obstruction:

In infancy: atresia of bile-ducts; chronic hepatitis; inspissated bile; cystic fibrosis of the pancreas.

In later childhood: malignant disease; hydatid cyst; ascaris infestation.

(*d*) With signs of infection:

In infancy: septicæmia; generalized virus infections; infective hepatitis; syphilis; toxoplasmosis.

In later childhood: infective hepatitis; acute yellow atrophy; homologous serum jaundice; Weil's disease; glandular fever.

(*e*) With failure to thrive:

In infancy: galactosæmia.

In later childhood: —.

(*f*) Certain poisons:

In infancy: —.

In later childhood: chloroform; phosphorus; chlorpromazine.

Jaundice with No Other Symptoms.

Physiological jaundice in the newborn is due in part—but only in part—to hæmolysis of excess red corpuscles. To a larger degree it is caused by functional immaturity of the liver in the first fortnight of life, reflected by poor capacity to excrete bilirubin and poor performance of other liver function tests. At birth the left lobe of the liver is about one-tenth larger than the right. Rapid involution follows withdrawal of the blood supply from the umbilical vein, so that for some days liver function is hampered by a steady decrease in the size of the organ.

Neonatal jaundice tends to be deeper in premature than in mature babies, and is occasionally severe enough to produce kernicterus, resulting in death or permanent brain damage. The reason for this intense jaundice is not at all clear. Kernicterus in premature babies seems to occur in some hospitals, but not in others. Mollison holds that "Synkavit" in doses above two milligrammes may be responsible.

Jaundice Associated with Hæmolytic Disease.

Jaundice associated with hæmolytic disease of the newborn is well known under the name of *icterus gravis neonatorum*. It should be remembered that sensitization of the mother to factors other than Rh antigen can occur. A B O incompatibility is the next commonest, the mother developing a high titre of anti-A or anti-B antigen. Jaundice in these cases is usually less serious than in Rh incompatibility. It has been asked why the jaundice appears so quickly after birth when it is not present before. The answer is surely that until the moment of birth bile pigment is excreted through the maternal circulation. There is little damage to liver cells in most cases, although it has been stated that Rh factor adheres to the baby's liver cells and is there attacked by maternal antigens. Brain damage, in the form of kernicterus, may well be a direct poisoning of certain brain cells by bilirubin when the blood level rises high enough. Experimental work on new-born kittens supports this. Serum bilirubin levels above 18 milligrammes per 100 millilitres are dangerous and should not be allowed to persist. Babies whose preliminary tests (hæmoglobin value, proportion of nucleated red cells, maternal antibody titre) have suggested severe trouble should have the serum bilirubin level estimated every twelve to twenty-four hours after the preliminary exchange transfusion. If it reaches 18 milligrammes per 100 millilitres a second exchange transfusion is performed, with the object of washing excess bilirubin out of the circulation. By this means the development of kernicterus can be almost entirely prevented.

Other forms of hæmolytic anæmia are exceptional in the newborn. In older children, acute hæmolytic anæmia (Lederer's), though rare, is quite dramatic. A child, previously in perfect health, develops in the space of two or three days intense pallor, fever, vomiting, moderately severe jaundice and hæmoglobinuria. Investigation shows a low red cell count with leucocytosis and large numbers of reticulocytes. Naphthalene poisoning may produce similar symptoms. As a rule a single blood transfusion proves curative.

Chronic hæmolytic anæmia (spherocytosis or acholuric jaundice) is much commoner. The child is more anæmic than jaundiced, and generally presents with lassitude, slight pallor and perhaps a palpable spleen. A yellow tinge of the conjunctiva is often just perceptible. Diagnosis rests mainly on the presence of anæmia with spherocytes, increased fragility, and a reticulocyte count above 5%, and is an indication for splenectomy.

Other causes of jaundice with anæmia in children are blackwater fever and reactions following incompatible blood transfusions. I have seen the latter in an Rh-negative hæmophiliac who had become unintentionally sensitized to Rh-positive blood.

[1] Read at the annual post-graduate course of FOCLA, Lismore, August 13 to 17, 1956.

Jaundice with Biliary Obstruction.

Jaundice with biliary obstruction must, in a newborn baby, at once arouse suspicion of atresia of the bile ducts. These babies attain an equilibrium after a while, excreting bile pigment in urine, tears and mucous glands. The jaundice is often not deep and the stools are not completely white, owing to admixture with bile-stained mucus. All these babies should be surgically explored, although in only about 20% is the obstruction outside the liver and thus operable.

Investigation by liver biopsy and post-mortem study of babies with obstructive jaundice showed that about 25% suffered not from congenital atresia, but from chronic hepatitis, presumably of virus origin (Gellis). Stokes, using on human volunteers the serum of the mother and of one such jaundiced baby, was able to produce jaundice in two out of five recipients of the baby's serum and in three out of five recipients of the mother's serum. The jaundice took seventy-five to one hundred days to develop. The baby later died of hepatic cirrhosis at the age of eighteen months. A repeat test on the mother three years afterwards again produced hepatitis in three out of five recipients of her serum. In a study of 42 babies with neonatal hepatitis, only one mother gave a history of the disease. The onset of the jaundice in the first fortnight of life, which happened in many cases, excluded infection with hepatitis A virus (infective hepatitis) acquired after birth. Transplacental infection with hepatitis A virus was ruled out, as the mothers had not suffered from this disease during the pregnancy.

Evidence favours the infection being hepatitis B (homologous serum jaundice), both from the long incubation period revealed in Stokes's experiments and from the known frequency of "silent" carriers. As these babies cannot readily be differentiated from those with atresia of the bile ducts, pale stools being common to both, exploration of the biliary passage and liver biopsy are necessary for diagnosis. Treatment is thereafter expectant, and about 70% recover. Two such cases have appeared at the Brisbane Children's Hospital within the last two years.

Obstructive jaundice from blocking of intrahepatic and extrahepatic ducts with inspissated bile is rarely seen nowadays. It was not infrequent in the days before the Rh factor was discovered, when it was known simply that blood transfusion sometimes helped babies with erythroblastosis. Excessive hæmolysis followed transfusion with Rh-positive blood, and the relatively insoluble bilirubin was deposited as a dark-green, gritty sludge. Sudden deepening of the jaundice and the appearance of white stools during a phase of hæmolysis arouse suspicion. If spontaneous recovery does not occur within two or three days, surgical exploration and irrigation of the ducts are required.

Biliary obstruction is a most unusual complication of cystic fibrosis of the pancreas, but it has been described and there has recently been an example at the Brisbane Children's Hospital. It is hard to understand why it does not occur more often, since inspissated mucus is responsible for the blocking of the ducts in both pancreas and liver. Treatment is unavailing.

Jaundice with signs of biliary obstruction in older children suggests malignant disease — probably lymphosarcoma or reticulosis of glands compressing the hepatic duct. Hydatid cyst is a very remote possibility, as the disease is now rare, and jaundice is not in any case a common symptom. Obstructive jaundice from the presence of an ascaris in the common duct is described, but is outside my experience.

Jaundice with Signs of Infection.

Jaundice in infancy accompanied by signs of acute infection, particularly vomiting and leucocytosis, generally indicates staphylococcal septicæmia. The organisms no doubt gain access to the blood-stream from the respiratory tract, but the presenting signs may be those of osteomyelitis or multiple abscesses. These babies are extremely ill, with high fever, vomiting, dehydration, rapid development of anæmia, enlargement of the liver and jaundice. The blood culture gives a positive result. The jaundice itself is due to swelling and necrosis of liver cells. Urgent treatment with "Aureomycin" or "Chloromycetin"—not penicillin — is necessary. It is important not to be misled by the jaundice into supposing that one is dealing with virus hepatitis, or by the liver enlargement into suspecting a localized abscess.

Direct liver infection by way of septic thrombosis in the umbilical vein is surprisingly rare.

Another rare but interesting type of jaundice in newborn babies is that accompanying generalized virus infections. This has been well described by Colebatch (1955). There are two varieties, cytomegalic inclusion disease and *herpes simplex* viræmia, both rather similar. The disease arises typically in premature or debilitated infants, and symptoms appear soon after birth, consisting of feeding difficulties, lethargy, vomiting, and jaundice. Respiratory distress and cyanosis are common, and there are likely to be hæmorrhages from the cord and mucosal surfaces and petechiæ in the skin. The liver and spleen are enlarged. Blood counts reveal anæmia, thrombocytopenia and erythroblasts. The prognosis is poor, the babies either dying or sustaining brain or visceral damage. Diagnostic findings are cytomegalic inclusion bodies in the cells of the urinary sediment, or *herpes simplex* lesions in the mouth and elsewhere.

True infective hepatitis is rare in infancy. Not being blood-borne, it must be acquired after birth, and cannot therefore in any case occur in newborn babies aged less than forty days.

Jaundice in congenital syphilis, though mentioned in the text-books, is a rare complication of a rare disease. I cannot remember encountering such a case.

Toxoplasmosis must not be forgotten. The usual symptoms in infancy are convulsions, hydrocephalus, chorioretinitis, other neurological and ocular signs and cerebral calcification. Jaundice is an inconstant symptom.

In later childhood, jaundice with signs of infection almost always means infective hepatitis, which year by year appears to be growing more common. The symptoms are well known, particularly the preliminary period of lassitude, anorexia and upper abdominal pain and tenderness. Biopsy studies have shown that liver damage persists long after the jaundice has faded. Rest in bed should be maintained for at least a week afterwards, and return to full activity should be very gradual.

Acute yellow atrophy, when not due to poisons or pregnancy, is probably a fulminating form of infective hepatitis. Certainly all intermediate grades are seen.

Homologous serum jaundice occurs occasionally 60 to 120 days after a blood or serum transfusion. The virus (hepatitis B) is not present in the fæces, as is that of infective hepatitis (hepatitis A) and there is no cross-immunity between the two infections. Symptomatically the two illnesses are almost identical.

Weil's disease follows immersion in water contaminated by the urine of infected rats, and appears after a ten-day incubation period. The chief symptoms are rigors, hæmorrhages, *herpes febrilis*, meningeal irritation, and renal damage leading perhaps to anuria. Jaundice appears only in the more severe cases. Leptospiræ may be discovered in the blood during the first six days by immediate guinea-pig inoculation. The results of agglutination tests become positive after ten days.

The majority (85%) of cases of glandular fever occur in children. The causal agent has not been isolated, but is probably a virus. Hepatic symptoms—enlargement of the liver and jaundice—are rare.

Jaundice with Failure to Thrive.

Jaundice with failure to thrive in early infancy suggests galactosæmia, a familial metabolic error characterized by jaundice, failure to thrive, enlargement of the liver, lamellar cataract and sugar in the urine. The underlying fault is a failure to form glucose from galactose, which

collects in the blood, where it acts as a poison, damaging liver cells and ultimately causing fatal cirrhosis. The urine contains a reducing substance which can be identified as galactose by the osazone test or by paper chromatography. It usually also contains albumin and amino acids. The glucose tolerance curve is normal, but the galactose tolerance curve remains high. The important point is that the condition is reversible if it is recognized early, and if the baby is kept on a lactose-free food, such as "Nutramigen", instead of milk.

Jaundice Due to Poisons.

Of the poisons which may cause jaundice from liver damage, chloroform and phosphorus are always mentioned, but are seldom seen. Chlorpromazine is a newcomer; jaundice occurs in a small proportion of cases when the dosage is heavy.

Reference.

COLEBATCH, J. H. (1955), "Clinical Picture of Severe Generalized Viral Infection in the Newborn", M. J. AUSTRALIA, 1: 377.

Reviews.

Quantitative Problems in Biochemistry. By Edwin A. Dawes, B.Sc., Ph.D., F.R.I.C., with a foreword by J. Norman Davidson, M.D., D.Sc., F.R.I.C., F.R.S.E.; 1956. Edinburgh and London: E. and S. Livingstone, Limited. 8½" x 5½", pp. 236. Price: 21s.

THIS little booklet will be of great help to senior students of biochemistry and to all workers in allied fields, particularly in medical research, who become interested in modern physico-chemical methods applied in biochemistry. In his foreword Professor Davidson points out that the present-day biochemist must have more than a nodding acquaintance with physical chemistry, and that the number of teachers of physical chemistry who have the necessary biological background to impart this knowledge is still all too small. While there is a number of books dealing with chemical calculations for students of chemistry, this is the first book dealing with biochemical problems in such a practical manner.

The minimum of fundamental information required for the solution of the problems is set out in eleven chapters, which deal with the determination of molecular weight (with particular stress on proteins); with acid-base relationships and electrolytic behaviour of amino acids and proteins; with thermodynamics (with a short discussion on the so-called "energy-rich bond"); with equilibria (activities, ionic strength); with reaction kinetics; with enzyme kinetics (Michaelis constant, enzyme inhibition, turnover number); with photometric analysis; with manometry; with bacterial growth; with oxidation-reduction potentials and potentiometry; and with the use of isotopes in biochemical work. In each of these chapters this theoretical introduction is followed by some examples of the relevant calculations, by references and suggested readings, and finally by a number of problems, the solutions of which are given at the end of the book.

One may quibble here and there at the essentialness of information given (for example, why introduce the antiquated "absorption ratio" in addition to extinction coefficient?). Nevertheless, the booklet will be welcomed by those who were not lucky enough to receive in their day such a valuable course of instruction as the one from which this book developed—more so, probably, than by those who now receive such a course.

Borderlands of the Normal and Early Pathologic in Skeletal Roentgenology. By Professor Dr. Alban Köhler; Tenth Edition, completely revised, with reference to illustrations and to text by Dozent Dr. E. A. Zimmer; English translation arranged and edited by James T. Case, M.D., D.M.R.E. (Cambridge); 1956. New York and London: Grune and Stratton, Incorporated. 10½" x 6¾", pp. 741, with 1300 illustrations. Price: $24.50.

THE work of Köhler has been looked upon for years as the most authoritative published anywhere in the world. The last English edition appeared in 1935 and is still treasured as a book of reference. The present edition is much enlarged, and the illustrations are more numerous and of higher quality. Every conceivable condition is dealt with in minutest detail; this is noticeable particularly in the discussion of the abnormal ossicles of the carpus and tarsus. Many unusual supernumerary ossicles are described and pictured. The developmental anomalies of the spine are many and varied, and the irregularity of the spinal epiphyses receives much attention; these latter are frequently subjects of argument in the law courts, and many patients undoubtedly receive compensation for irregularities shown in X-ray films which are really slight departures from the normal. The various conditions described by the authors carry references to the original articles in which they have appeared, and an extensive bibliography is one of the features of the work. The various paragraphs are packed with detailed information and need careful attention when they are read. A feature of the work is the amount of personal experience brought into it. After reading the book one realizes with humility how little one knows of this vast subject. The book can be highly recommended as a book of reliable information and worthy of frequent reference.

Women of Forty: The Menopausal Syndrome. By M. E. Landau; 1956. London: Faber and Faber, Limited. 7½" x 5", pp. 49. Price: 5s.

A COMMONPLACE of medical practice is the frequency with which patients demand to know why they experience certain symptoms. They often seem more interested in probing the cause of their complaint than in receiving treatment directed towards its cure. An old doctor of our acquaintance used to answer the query: "What is this medicine for, doctor?" with the terse reply: "For you." Few of us can get away with such firm brevity, and often we have to spend time trying to give instructive little talks on physiology or pathology to eager but untrained ears.

The menopause is one subject on which some women really do need our help in explaining symptoms and dispelling fear, and the talk is usually more helpful than the sedatives or hormones which may or may not be prescribed. Dr. Landau's little book, "Women of Forty", can be recommended to any woman who wishes to read a short and authoritative description of the menopause. The physiology of the menopause and the symptoms which may occur are simply and well explained. Disturbances of mood are discussed and good advice is given on their control, together, with a sensible discussion of occupation and employment inside and outside the home.

With regard to physical treatment, the author confines herself to remarks on rest and exercise and attention to diet to avoid obesity. She points out that medication, necessary for the minority of women, must be taken only on the advice of their doctor.

Special Lectures in Biochemistry, 1954–1955, given at the Department of Biochemistry, University College, London. London: H. K. Lewis and Company, Limited. 11" x 8½", pp. 106, with 36 illustrations. Price: 12s. 6d.

"IT is the custom of University College London to invite eminent scientists, classical scholars, historians and others, to deliver special so-called public lectures from time to time." The publication "Special Lectures in Biochemistry" gives a selection of papers which have been given on biochemical subjects during 1954-1955.

The first paper is "The Relationship of Structure to Catalytic Activity of Vitamin B6", by E. E. Snell. Then follow three papers on the respiratory mechanisms of bacteria by S. R. Elsden. R. J. P. Williams gave four lectures on "Metal Complexes as Models for Metallo-Enzymes". Finally there is a "Colloquium on Biochemical Aspects of Chelation", eight authors giving seven papers.

As will be seen by the titles of the papers submitted, all the lecturers dealt with very advanced biochemistry. To follow adequately any of the lectures would require a considerable knowledge of advanced biochemistry and organic chemistry. The collection of lectures can be recommended to research workers in the different fields dealt with as presenting adequate and detailed accounts of the present knowledge.

Clinical Laboratory Methods and Diagnosis: A Textbook on Laboratory Procedures with their Interpretation. By R. B. H. Gradwohl, M.D., D.Sc.; Fifth Edition, Volumes I and II; 1956. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical), Limited. 10" x 7", pp. 2791, with illustrations. Price: £21 3s. 6d.

SINCE the last edition in 1949, this veritable encyclopædia of laboratory diagnosis has been extensively revised. Obsolete procedures have given way to the latest, and many new techniques in diagnosis have been inserted. In particular the following may be cited: electrophoresis, flame photometry,

spectrophotometric methods, tests for the diagnosis of coagulation defects, blood groups and transfusion problems and frozen section preparations.

The book is in two volumes. All phases of laboratory diagnosis are effectually covered. Briefly, sections deal with the types, uses and care of laboratory apparatus; the chemical examination of body fluids; hæmatological investigations, with a chapter on the blood groups and transfusion; medical bacteriology and mycology; parasitology and some aspects of tropical medicine and serology. Further, there are chapters dealing with the preparation of histological sections, toxicology and forensic medicine, basal metabolism and electrocardiography.

Although this is chiefly a reference book on the methods of laboratory diagnosis of disease, most sections also include an introduction and a few paragraphs on the interpretation of results. Accepted laboratory methods are set out with extreme clarity, conciseness and completeness. Small points in finesse of technique with detailed procedures for the solution of difficulties encountered are of great assistance.

The book is well illustrated, particularly in the hæmatological and parasitological sections.

To sum up, it may be emphasized that, covering such a large field of work, this is a surprisingly up-to-date book and would allow a worker to dispense with several smaller volumes dealing with individual phases of laboratory work.

Ciba Foundation Symposium on Bone Structure and Metabolism. Edited by G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., and Cecilia M. O'Connor, B.Sc.; 1956. London: J. and A. Churchill, Limited. 8¼" x 5¼", pp. 310, with 121 illustrations. Price: 45s.

THE latest Ciba Foundation Symposium to be published is on bone structure and metabolism. Twenty-eight specialists from Europe and America met in London in July, 1955, and produced twenty papers with discussions on various aspects of structure of bone, pathological conditions in bones and the effect of radiation, and the dynamics of bone organization including the biochemistry of bone formation. The first four papers deal with the anatomical and molecular structure of bone, bone salts and the matrix of bone.

The relation of citrate to bone formation is discussed, as also are the effects of magnesium deficiency in stock. There are studies on the repair of fractures by the use of P^{32}. There are two papers on vitamin *D* and its action in bone formation, but there is little new in these, the final statement being that the absorption of calcium from the small intestines is increased by vitamin *D* throughout life, and that the defective accumulation of bone salts is chiefly due to defective absorption.

The relation of parathyroid secretion to bone formation is considered.

There are several papers on pathological studies on bone and the important part played by bone in the storage of radioactive materials.

As always, the discussions are almost more valuable than the papers.

This is a good example of the Ciba Foundation Symposia, and while it will be particularly valuable to the orthopædist, most medical men will find much of interest in the volume.

The Year Book of Medicine. Edited by Paul B. Beeson, M.D., Carl Muschenheim, M.D.; William B. Castle, M.D., Tinsley R. Harrison, M.D., Franz J. Inglefinger, M.D., and Philip K. Bondy, M.D.; 1956-1957 Year Book Series; 1956. Chicago: The Year Book Publishers, Incorporated. 8" x 5¼", pp. 744, with illustrations. Price: $6:75.

THIS is one of the most comprehensive of the Year Books, covering as it does a wide range of world medical literature. Its six editors are the same as for the two previous volumes. Part I deals with infections and has sections on the general chemotherapy of infection, on chemoprophylaxis of infection, on Candida infections following the use of antibiotics and cortisone, on cortisone, on various particular kinds of infections grouped according to the organism, the site or the specific condition, on virus tissue culture in clinical diagnosis, on "new" viruses, on "collagen" diseases, on diseases of uncertain ætiology, on nervous system manifestations, and on "fascinating names". Part II, which deals with the chest, has sections on anatomy, physiology, emphysema, the Hamman-Rich syndrome, Wegener's granulomatosis and *periarteritis nodosa*, pneumonokoniosis, neoplasms, lung abscess, mycoses, pleurisy and pneumothorax, and tuberculosis. The blood and blood-forming organs occupy Part III, which has sections dealing with the usual hæmatological disorders; blood dyscrasias associated with the administration of drugs receive special mention. The sections of Part IV, which is devoted to the heart and blood vessels and the kidney, refer to congenital heart disease, rheumatic heart disease and valvular disease, hypertension, coronary disease, arrhythmias, electrocardiography, pathological physiology, cerebral and peripheral vascular disease, and the kidney. The material in Part V, which is concerned with the digestive system, is subdivided on an anatomical basis. Metabolism is the subject of Part VI, which has sections on the adrenal glands, the thyroid gland, carbohydrate metabolism, calcium, phosphorus and the parathyroid glands, the pituitary gland, growth, weight and nutrition, metabolic diseases and lipid metabolism.

My Story: Memoirs of a New Zealand Nurse. By Mary I. Lambie, C.B.E., with a foreword by Michael H. Watt, C.B.E., M.D., D.P.H., F.R.C.P.; 1956 Christchurch: N M. Peryer, Limited 9" x 6¾", pp. 100, with illustrations. Price: 30s.

THE curse of most books of memoirs is prolixity, and few writers in this *genre* can compress their experiences and opinions into a sizable compass. This story of Miss Mary Lambie's life and career is interwoven with a detailed account of the development of the nursing service in New Zealand from the time when she began her training in Christchurch in 1910. Becoming engaged in the organization of "post-graduate" training of nurses, she was in 1931 appointed Director of the Division of Nursing of the Health Department in the Dominion. It is clear that she merited the recognition accorded her for this work, and for her contribution to the development of the nursing services in Fiji and the islands of the South Pacific when these came under the control of the New Zealand Department of Health. For all interested in the history of nursing this book will have value as a record, in spite of the unessential detail which clutters many of its pages.

Gynæcology. By Douglas H. Macleod, M.S. (London), F.R.C.P. (London), F.R.C.S. (England), F.R.C.O.G., and Charles D. Read, M.B. (New Zealand), FR.C.S. (England and Edinburgh), F.R.A.C.S., F.R.C.O.G., with a section on anatomy by James Snyder, M.D., F.R.C.S., and a section on physiology and endocrinology by Russell Fraser, M.D., F.R.C.P.; Fifth Edition; 1955. London: J. and A. Churchill, Limited. 10" x 6¾", pp. 876, with 551 illustrations, 27 in colour. Price: 80s.

THE fifth edition of this book maintains the high standard established by the original authors, Eden and Lockyer, and sustained by Sir Beckwith Whitehouse in the fourth edition.

In spite of an interval of twenty years between the appearances of the fourth and fifth editions, the character of the book has been preserved and a pleasing sense of familiarity is imparted to the reader acquainted with the earlier editions. The subject matter is well presented, and the book is printed on high quality glossy paper. The illustrations are adequate in number and of excellent quality.

It is rather surprising to find, in an English text-book, the plural of the words vulva, *nullipara* and *multipara* employed on occasions where the singular should apply, and vice versa in some instances. These errors occur in the otherwise excellent first chapter. Throughout the text of the book a few relatively minor but aggravating errors occur, which might have been avoided by careful proof reading.

The description of the investigation of the female partner in infertility does not include culdoscopy.

As in previous editions, the section on *leucoplakia vulvæ* and kraurosis is excellent. A clear exposition is given of a subject on which the writings of many authors have been confusing and confused.

A notable omission in the description of the treatment of *pruritus vulvæ* is the local application of hydrocortisone ointments or lotions.

In the description of the treatment of vulval epithelioma on page 314, the all-important deep femoral lymph nodes (including the node of Cloquet) are omitted from the list of lymph nodes to be removed. This group of nodes, however, receives adequate treatment in a later section on vulval and vaginal operations.

In the sections dealing with the clinical signs of cancer of the vaginal portion of the cervix and with the diagnosis of morbid conditions of the cervix, no mention is made of colposcopy, although the colposcope is described briefly in an earlier section on methods of examination.

In the description of salpingostomy and tubal grafting the use of polyethylene tubing is not mentioned.

In the section dealing with post-operative complications, when the treatment of mechanical intestinal obstruction is described, the advice given is that "when a diagnosis of obstruction has been made laparotomy should be immediately undertaken while the patient is in a fit condition to stand the operation, and the cause dealt with. If fæcal vomiting is present the stomach should be emptied and thoroughly irrigated through a stomach tube . . ." Such pre-operative treatment is regarded as quite inadequate and not in line with modern teaching and practice, which advocates gastric, gastro-duodenal or gastro-intestinal aspiration decompression with replacement of and maintenance of fluid and electrolyte balance as necessary preliminaries to the relief of obstruction by operation and as essential post-operative treatment.

Few surgeons would agree with the advice that "purgatives, colon irrigation by high enemata and the use of certain drugs such as acetylcholine and prostigmine which act powerfully upon intestinal peristalsis are the methods of choice" in the treatment of paralytic ileus. Gastro-intestinal suction is advocated by the authors in severe cases with failure to respond to the earlier methods, and they recommend that the intestinal contents which have been removed by continuous aspiration should be returned to the body in the form of a nutrient enema.

The continuous intravenous use of saline is mentioned in the treatment of peritonitis and paralytic ileus, but no efficient method of replacement and maintenance of fluid and electrolyte balance is described.

Pulmonary embolism and infarction are described briefly. The paragraph describing pulmonary infarction is unaltered from the third (1928) edition, and states that "when a minute branch of the pulmonary vein becomes blocked by a portion of clot carried in the blood stream the result may be the formation of a patch of consolidation, i.e. an infarct". It is unfortunate that such a statement should have appeared in three successive editions of this work.

In spite of these few criticisms, this book is strongly recommended as an authoritative work of a very high standard. The authors are to be congratulated on its quality, and it is hoped that the sixth will follow the current edition at an interval considerably shorter than that between the fourth and fifth editions.

Medical Parasitology: For Medical Students and Practising Physicians. By William G. Sawitz, M.D.; Second Edition; 1956. New York, Toronto, London: McGraw-Hill Book Company, Incorporated. 9″ × 6″, pp. 351, with illustrations. Price: $6.00.

"MEDICAL PARASITOLOGY", by William G. Rowitz, has reached its second edition. It is intended for medical students and practising physicians, and the manner in which it is set out and illustrated is ideally suited to its purpose. There are tables and definitions which show, economically yet amply, the systematic relationships of the species and the terminology used to describe them and their activities, while a useful vocabulary of technical terms is included.

A typical section covers, concisely and lucidly, with illustrations which really illustrate, Acarina, differentiation of ticks and mites, classification of ticks, mouth parts of ticks, ticks of medical importance in the United States, pathogens transmitted to man by ticks, treatment and prevention—all in seven and a half pages. The author achieves this by omitting unnecessary detail, avoiding padding, and restricting the scope almost exclusively to American parasites and diseases; *Rhipicephalus sanguineus*, *Ixodes* spp., and *Ornithodorus moubata*, with the diseases they carry, receive only a bare mention, while tick typhus does not even receive any mention at all. This is not detrimental—the American matter is well covered and gives a sound basis from which anybody working in other countries can soon extend his local knowledge. To have included the innumerable species of the rest of the world, in view of the declared scope of the book, would have resulted in overloading it and reducing its value.

New Pathways in Cellular Pathology. By Gordon Roy Cameron, M.B., D.Sc. (Melb.), F.R.C.P., F.R.S.; 1956. London: Edward Arnold (Publishers), Limited. 8½″ × 5½″, pp. 98. Price: 16s.

MORPHOLOGICAL pathology is the study of tissues affected by disease, and to a certain extent the processes of disease. Certain appearances in cells are usually described as toxic, and we ascribe them to altered metabolism. These terms are convenient, but they explain little. Meanwhile, biochemists and biophysicists with their new techniques have been discovering the structure and organization within the cell, so that a new morphology is arising and with it a new physiology and biochemistry. One can state that there is already a morphology of metabolism. Accordingly there must be an interpreter who can correlate the alterations in enzyme systems with the manifestations of disease. In this slim volume, which is expanded from lectures given in Leeds and Dublin during 1954 and 1955, Professor Cameron gives an outline of the new methods of investigating altered function of the cell. He describes how each component, the cell surface, the cytoplasm, the nucleus, the mitochondria and other minute structures, can be examined, and illustrates the procedures mainly from work performed in his own laboratory. This book points the way in which pathology is progressing, and it should be read by all those who are dissatisfied with the limitations of morphological pathology.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"The Year Book of Obstetrics and Gynecology (1956-1957 Year Book Series)", edited by J. P. Greenhill, B.S., M.D., F.A.C.S., F.I.C.S. (Honorary); 1956. Chicago: The Year Book Publishers, Incorporated. 7½″ × 5″, pp. 592, with 82 illustrations. Price: $6.75.

One of the Practical Medicine Series of Year Books.

"Practical Pediatric Dermatology", by Morris Leider, M.D.; 1956. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical), Limited. 10″ × 6¾″, pp. 433, with 280 photographs and 13 drawings. Price; £5 15s. 6d.

Designed for general practitioners and pædiatricians as well as for dermatologists.

"Sex in Christianity and Psychoanalysis", by William Graham Cole; 1956. London: George Allen and Unwin, Limited. 8½″ × 5½″, pp. 343. Price: 21s.

The author presents the interpretations of sex put forward by leading Christian figures and institutions, and by Freud and contemporary psychoanalysts, and then his own "critical reconstruction of christian interpretations of sex".

"The Year Book of Pediatrics (1956-1957 Series)", edited by Sydney S. Gellis, M.D.; 1956. Chicago: The Year Book Publishers, Incorporated. 7¾″ × 5¼″, pp. 480, with illustrations. Price: $6.50.

One of the Practical Medicine Series of Year Books.

"Athletic Injuries: Prevention, Diagnosis and Treatment", by Augustus Thorndike, M.D.; Fourth Edition, thoroughly revised; 1956. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 8¼″ × 5½″, pp. 252, with 113 illustrations. Price: 49s. 6d.

Written from experience associated with American college sport.

"The Care of the Expectant Mother", by Josephine Barnes, M.A., D.M. (Oxon.), M.R.C.P. (London), F.R.C.S. (England), F.R.C.O.G.; 1956. London: Pitman Medical Publishing Company, Limited. 9″ × 5¾″, pp. 280, with illustrations. Price: 30s.

Written for all concerned in the management of pregnancy, from medical students to obstetrics consultants.

"Practical Forensic Medicine", by Francis E. Camps, M.D., and W. B. Purchase, C.B.E., M.C., M.B., D.P.H., with a foreword by H. Edmund Davies, LL.B., B.C.L.; 1956. London: Hutchinson's Medical Publications, Limited. 9¼″ × 6¼″, pp. 541, with many illustrations. Price: 75s.

The authors have attempted to bring the subject up to date.

"The Sexual, Marital and Family Relationships of the English Woman", by Dr. Eustace Chesser, in collaboration with Joan Maizels, Leonard Jones, B.Sc., Ph.D., Brian Emmett, B.Sc., and with the assistance of Professor F. A. E. Crew, F.R.S., Professor Alexander Kennedy, M.D., D.P.M., Kenneth Walker, M.A., M.D., F.R.C.S., I.C.S., Doris Odlum, M.A., M.D., M.R.C.S., I.C.S., D.P.M., and Canon Hugh Warner, M.A.; 1956. London: Hutchinson's Medical Publications, Limited. 9¼″ × 6¼″, pp. 680, with many tables and charts. Price: 75s.

Based on *questionnaires* completed by some 6000 married and single women approached by their general practitioners.

The Medical Journal of Australia

SATURDAY, MARCH 9, 1957.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

MINIMAL PULMONARY TUBERCULOSIS DISCOVERED BY MASS RADIOGRAPHY.

IN his preface to the first Prophit Survey Report, published in 1948, Lord Moran indicated that the generous legacy received under the will of the late Mr. J. M. G. Prophit would allow "a large scale investigation into the natural history and epidemiology of tuberculosis in the young adult population". "The main object of the Survey", wrote Lord Moran, "was to try to determine whether it is possible to pick out those persons or groups of persons most likely to develop tuberculosis." Happily, the work of the Prophit Tuberculosis Survey did not end with that investigation and report. It was realized at the time that the interpretation of many X-ray appearances was uncertain, and was likely to be clarified only by observing the evolution of the lesions over a prolonged period. With increasing use of mass radiography, the demonstration of small shadows in apparently healthy people became very frequent. It was therefore resolved to make a careful assessment of at least 500 people of each sex found to have abnormalities of this type and to observe them over a five-year period. Following on this the Royal College of Physicians has now published its second Prophit Report, entitled "Minimal Pulmonary Tuberculosis Discovered by Mass Radiography (Fluorography)".[1] The Prophit Scholar, V. H. Springett, here records the method of selection, initial examination and periodic reexamination of 1213 persons, aged fifteen to forty-four years, accepted into the survey. The patients had attended voluntarily at either of two mass radiography units in London. The miniature films had been read once; and whenever an abnormality was reported, the subject was invited to return for a full-sized direct radiograph. Springett attended the sessions at which these large films were read. Patients selected for further observation were those whose radiographs showed small lesions of several types without cavitation, pleural effusion or other pulmonary disease. The initial clinical examination included the recording of history and physical findings, tuberculin testing, and the taking of two laryngeal swabs for bacteriological examination and a sample of blood for measuring the erythrocyte sedimentation rate. Patients who could produce sputum were given a sterile container in which to submit a specimen. An interesting point in history-taking was the clear distinction between symptoms volunteered by the patient and those later admitted in answer to direct questions. Subsequent examinations took place at intervals of three months, six months, one year, eighteen months, two years, three years, four years and five years. At each of these visits the initial examinations and tests were repeated, and postero-anterior and antero-posterior radiographs of the chest were taken. Many patients required treatment during the survey. Specific indications were laid down; and whenever these were found, patients were referred through their own doctors, generally to local chest physicians or clinics. Actual decisions on treatment were made by the latter, and Springett maintained contact with this group of patients by visiting the various clinics concerned.

The results of the survey have been most carefully classified and presented. The reason for a detailed classification of material such as this is to permit the separation of those characteristics or groups of characteristics which are associated with or give warning of progression of disease from those for which no such significance is demonstrated. Thus it may be possible to separate small lesions found by mass radiography into a "low risk" and a "high risk" group, or into a group requiring observation alone and one in which treatment is, or is likely to be, required. With this general aim the initial radiographic appearances were classified in respect of type, size and location of the abnormality. In order to assure constancy of interpretation, films were classified more than once, and in cases of doubt Springett had the assistance of two eminent radiologists, both members of the Prophit Committee, Dr. L. G. Blair and Dr. Peter Kerley. Changes in radiographic appearance were assessed by three independent observers without clinical or bacteriological information. Differences of opinion were settled at committee sessions. The method of selection of these patients did not allow conclusions to be drawn as to the prevalence of various types of lesions in London. However, two interesting observations were that the yield of minimal lesions declined with increasing age and was consistently, though not significantly, higher in women than in men. The common type of abnormality consisted of foci two to ten millimetres in diameter in clusters. These were present in over 80% of cases and were the only abnormalities in over 50%. In almost a quarter rounded homogeneous shadows were seen. The lesions were of an average size of 10 to 15 square centimetres, and three-quarters of them were confined to the upper zone.

[1] "Minimal Pulmonary Tuberculosis Found by Mass Radiography (Fluorography)", a report to the Prophit Committee of the Royal College of Physicians by V. H. Springett, M.D., M.R.C.P., and including results of work done by A. J. Eley, M.A., M.B., B.S., D.M.R.(D.); 1956. London: H. K. Lewis and Company, Limited. 10" × 7½", pp. 247, with illustrations. Price: £2 2s.

Homogeneous round foci were generally smaller and more widely scattered than other types of lesion. About 7% showed calcified primary foci. Nearly half the patients had previously been examined by chest radiography, and in just over one-quarter of these the previous X-ray finding was confirmed as normal. Few patients volunteered that they had any symptoms, but on direct questioning about one-third admitted to having cough and sputum; and dyspnœa and chest pain were admitted by almost 20%. Hæmoptysis was admitted by 2·1%. The vast majority had no abnormal physical signs, but the 1% with definite abnormal signs suggestive of activity all had symptoms and positive bacteriological findings. The results of tuberculin tests were positive in 99% of cases, but the level of sensitivity appeared to bear no relation to activity of disease. In persons from whom both sputum and laryngeal swabs were examined, the findings on sputum examination were more often positive. However, in the whole group more positive results were obtained from laryngeal swabs than from sputum. The likelihood of positive bacteriological results was related directly to the size of the lesion, but lesions showing calcification or linear shadows gave fewer positive results.

Thirty-two patients sought advice between routine visits because of symptoms. Hæmoptysis, chest pain, sweating and fever appeared to be more frequently associated with objective evidence of activity than did respiratory infection, faintness and lassitude. Cough and chest pain became less frequent as the survey proceeded. Symptoms admitted in answer to a direct query were not associated with objective signs of activity. Crepitations were heard very infrequently (in less than 2% of patients examined at any time up to five years); but when they were heard there were always other evidences of activity, and this despite Springett's later statement that neither crepitations nor rhonchi show any strong association with objective signs of activity. It is comforting to those who still like to use the stethoscope in examining the tuberculous to know that, though crepitations were rarely heard in these patients with minimal lesions, they were, when present, a sure indication of activity. Almost 30% of patients showed radiographic evidence of worsening within five years. In the vast majority of these the worsening occurred in or near a preexisting lesion, and in the first six or twelve months after the lesion was discovered. Positive bacteriological results were also found in 30%, and these, too, occurred overwhelmingly in the first year. Most patients gained weight during the survey. The erythrocyte sedimentation rate was raised in a significant proportion of cases at the time of demonstration of activity. Tuberculin sensitivity increased during the first year of observation and then usually returned to its initial level. It was not shown to be related to evidence of activity. Of those in whom either tubercle bacilli or radiographic worsening were found, about half showed both, usually within a few months of each other. During the first year of observation tubercle bacilli usually appeared before radiographic deterioration. In later years this was reversed. Initial findings associated with a greater likelihood of radiographic deterioration or isolation of tubercle bacilli were a relatively large lesion, absence of calcification or linear shadowing, isolation of tubercle bacilli from sputum or laryngeal swab, age less than thirty years, a normal radiograph within the past five years, recent family contact with tuberculosis, and the presence of symptoms or physical signs. It is interesting to note that single persons had a worse prognosis than married; and apical localization on the initial radiograph was a favourable feature. These are beautiful examples of indirect associations which, to the uncritical observer, might be quite misleading. Springett points out that the single group have a worse prognosis because they are younger, and prognosis is clearly demonstrated as being related to age. Similarly, apical lesions are on average smaller, and the association of size of lesion with subsequent fate is clearly shown. No association is demonstrated in this survey between five-year progress and any of the following features: occupation, hours of work, and travelling time; economic and housing conditions; history of bronchitis, pneumonia and pleurisy; erythrocyte sedimentation rate; level of tuberculin sensitivity. Here it must be borne in mind that the group from which these patients were selected was by no means a cross-section of London's population, but was made up mainly of civil servants and office workers. They represent a rather homogeneous social and economic group, with no wide diversity of occupations or working conditions.

The chief indications for treatment were radiographic deterioration and isolation of tubercle bacilli. When only one of these features occurred, treatment was often delayed or not given. In many of these cases no further evidence of activity occurred during the survey period. It must be remembered, however, that in the early years of the survey chemotherapy was not available, and even in the final stages it was by no means universally used for active lesions of minimal extent. Rest and collapse therapy were the major means of treatment. About 8% of those treated ultimately underwent major operations. Almost two-thirds of these patients lost no time from work because of tuberculosis. Half of the remainder were treated and returned to work within the five-year period. The number of those who died was less than the expected number of deaths in a population of the age-sex distribution of the survey group.

In his lucid general summary and discussion Springett confirms and emphasizes the finding of Mitchell, Reisner and Downes, Lincoln, and others, that the risk of any evidence of activity first appearing becomes less as the period of observation without evidence of activity increases. Hence the need for frequent examination in the years immediately succeeding the appearance or discovery of a lesion, with lengthening intervals in later years. After three years the risk of breakdown has become fairly small and constant at a level probably rather greater than might be expected in a group with normal chest radiographs. As a corollary of this, patients who had had a normal radiograph shortly before the discovery of the lesion showed the greatest tendency to progress. From these observations it is reasonable to conclude that persons with minimal lesions require some supervision over many years; and that the great importance of new lesions is a sound reason for making repeated surveys of the same population. The extent to which minimal tuberculosis interfered with normal life was considerable. One-third of the subjects required treatment, and over half of these were in hospital for more than a year. With present methods of treatment there is almost certainly some improvement in this regard.

Finally, Springett discusses the management of minimal tuberculosis. The important decision between treatment

and observation depends upon the summation of the various factors which have been studied in this survey: age and sex; radiographic appearance, especially size of lesion, and presence or absence of calcification; a knowledge of symptoms volunteered by the patient; recent pleurisy or tuberculosis in the family or associates. All these are available at the first examination. Within a few days there will also be available the results of microscopic examination of sputum (if any), and of erythrocyte sedimentation rate and tuberculin tests, and it should be possible to learn results of any previous radiographs or tuberculin tests. These are, of course, many of the criteria that chest physicians have been using for a long time in deciding between observation and treatment of the individual with minimal tuberculosis. But the great merit of this survey is that it provides convincing evidence of the relative importance of these factors and the relative unimportance of others on which perhaps too much reliance has been placed in the past. The decision between observation and treatment for minimal lesions has always been of great importance to patient and physician. To the patient the choice lies between temporary dislocation of normal life now in an attempt to avoid more serious dislocation later, and continuance of normal activity with acceptance of the attendant risk. Springett has demonstrated that serious progression without warning was extremely rare in his group observed according to a definite plan. The advent of effective chemotherapy has made the decision between observation and treatment less urgent, but it has not made it any easier. Treatment of the initial lesion is simple and safe, but so, too, is treatment of any extension which may occur during observation. Springett's observations have, however, enabled him to suggest a rational basis for making the decision between observation and immediate treatment almost as soon as the lesion has been discovered. Lesions in young persons, extensive lesions, fresh lesions of any size, lesions without calcification—these are the groups in which progression of disease is likely to occur, and in which early treatment is indicated.

While the clinical and radiological indications for treatment have been logically and convincingly deduced from the observations made, the bacteriological indications leave one a little uneasy. In Springett's view, the recovery of tubercle bacilli from a person with a minimal lesion is not of itself an absolute indication for treatment. Even if we allow that his patients were apparently healthy adults, generally remarkably free from symptoms or other evidence of disease, we cannot accept the statement that "as results of sputum and other bacteriological tests become available, it will be necessary to advise treatment *to most of those* giving a positive result". Surely, if the eradication of tuberculosis is one of our aims, then the tubercle bacillus must be treated as the Army treats its enemy—it must be "sought out and destroyed wherever he may be found".

Two other features of this report call for comment. In the first Prophit Report tables occurred throughout the text. This time they are consolidated in the rear pages, where they are in danger of neglect. The second feature is the nature and quality of the radiographic reproductions. In many leading journals and in almost every text-book and monograph, radiographs are reproduced as negatives—that is, in the same phase as the direct radiograph. In both Prophit Reports we find them reproduced as positives, in the reversed phase. Few people are accustomed to viewing this type of reproduction. When, in addition, the reproductions show too much contrast—the blacks too black and the whites too white—they become well-nigh useless. Fortunately, Springett writes so explicitly, and defines his terms and describes appearances so lucidly, that one can follow the whole argument without adequate visual aids.

This report contains a wealth of relevant fact and soundly based opinion. The first chapter sets out admirably the plan for conducting a long-term clinical observation. In the two final chapters the findings are summed up, discussed and evaluated, and a rational system for the management of minimal tuberculosis is presented. In many parts of this country patients with minimal tuberculous lesions are referred by mass radiography organizations to private practitioners for initial assessment. Many of the facts which Springett has found important are available to those interpreting the films. Reports and advice which they give should be framed in the light of Springett's findings. The family doctor will be in possession of many facts not available to the mass radiography staff. If he is aware of the relative importance of these facts, as demonstrated by Springett's careful investigation, he will give his patients better service.

Current Comment.

THE PROGNOSIS IN SYPHILITIC AORTIC INCOMPETENCE.

ULTIMATELY progress in the management of any condition must be assessed in the light of the natural history of that disease, and it is one of the many paradoxes of medicine that it is sometimes not until some more or less useful form of treatment is available that the necessity for a standard of reference impels someone to collect and analyse the necessary data. By this time the analysis must inevitably be retrospective, and interpretation of the results must be made with caution. Of course, there are many difficulties in the way of effective anterospective studies, especially in the less common conditions, but such a project would be well worth the time of enthusiastic younger men with sufficient patience to wait a decade or so for their results to approach the stage of publication. Point is given to these reflections by the review of the literature relating to prognosis in aortic incompetence of syphilitic origin which is appended to their own study by J. C. Leonard and W. G. Smith.[1] Thus, after careful assessment of the contributions of a number of workers over thirty years, it is possible for them to conclude that the effectiveness of arsenic and heavy metal treatment has never been universally accepted, which is tantamount to saying that benefit has never been adequately demonstrated. Is it that a remnant of the therapeutic enthusiasm of our ancestors lives on in the age of the controlled experiment, or is it that the problem of the appropriate treatment of a disease once not uncommon was too difficult to solve? On past performances, in another thirty years the same indecision will exist in regard to penicillin therapy; but perhaps the account of the natural history of the condition given by Leonard and Smith will be considered an appropriate standard for comparison, or perhaps the disease will have been forgotten by all but the standard text-books. Certainly, Leonard and Smith's series of 374 cases, gathered mainly from London teaching hospitals, is an impressive one, and on the whole the

[1] *Lancet*, February 2, 1957.

material is well presented. Only 13% of the patients remained untraced, although the period covered was thirty years; as all the deaths, or most of them, were traced, it must be assumed that many of the untraced group are alive.

Leonard and Smith review their data from a host of different points of view, and it is impossible to present all the information in the present abstract. The material selected for comment is that which seems most likely to be statistically valid, but it is a minor criticism of the paper that the authors quote ranges rather than standard deviations (the clinical interest often pertaining to the latter does not justify this omission), and that important deductions are based upon differences which the reader must assume to be statistically significant. About a third of the patients gave a past history of venereal disease, a mean of thirty-one years prior to the diagnosis of aortic incompetence. About two-thirds complained of dyspnœa on exertion and/or paroxysmal nocturnal dyspnœa, and half of cardiac pain; 13% were free of symptoms. Loss of weight was prominent in about 18 patients, and this seemed to carry a poor prognosis. Of interest in regard to the physical signs was the finding of a systolic thrill in patients without either stenosis of the valve or aneurysm of the aorta at autopsy. Arrhythmias were uncommon, about 7% of patients, notably in older age groups, developing auricular fibrillation. Complete heart block occurred in only three cases.

The results of special investigations were much as might have been anticipated, the Wassermann reaction being positive in 96%. The mean erythrocyte sedimentation rate was 27 millimetres in the first hour in the 143 cases in which it was performed, and this test result remained, on the average, high even in the presence of congestive cardiac failure. There was evidence that a high level was associated with a less satisfactory prognosis. Electrocardiograms showed signs of left ventricular hypertrophy or strain in about 75% of cases, while 15% were normal (the figures suggest that the latter proportion might have been smaller if more of the patients had had more than the three standard leads taken). Two hundred and twenty-two patients died, and of these, the deaths of 207 were attributed to cardio-vascular disease. Of 60 patients who came to necropsy, one was found to have bacterial endocarditis, a condition which had been suspected in two others of this group.

The authors' review of the factors affecting survival after diagnosis is worth study by those particularly concerned with this disorder. About two-thirds survived two years, but the five-year and ten-year survival rates were 45% and 25% for males and 65% and 55% (approximately) for females. At the end of ten years after diagnosis, 65% of patients under the age of thirty-nine years at diagnosis were alive, compared with less than half those at diagnosis between the ages of forty and forty-nine years. Nearly three-quarters of the symptom-free patients at the time of diagnosis were alive after ten years; the mean age of these patients at diagnosis was fifty-two years, and most of them were men. Table IV in this paper, relating to the age and sex of patients presenting with cardiac pain, is distinctly misleading in that the ages twenty to forty-nine years are bracketed, the next age group being fifty to fifty-nine years; such a procedure tends to over-emphasize the frequency of the symptom in the younger age groups; what the percentage figures in Tables IV and V refer to is not clear. At all events, Leonard and Smith make out a good case that anginal pain is not associated with a particularly poor prognosis; the mean survival of the patients who died having suffered from this symptom was about six years from the time of its onset, and this is an overall under-estimate, as some patients are still alive. Nearly 40% of patients with angina lived for ten years. Heart failure, which is commoner in older age groups, is of much worse prognostic significance, as half of the patients were dead in a year and two-thirds in two years, the mean survival of those who died being only eighteen months.

Leonard and Smith have paid special attention to an analysis of their data in terms of the effects of treatment with bismuth and arsenic and with penicillin, as compared with one group of their patients who had had "inadequate treatment". The details of their calculations need not concern us here, but they appear to indicate that either main form of treatment is unequivocally superior to little or no treatment; the survival is about five years with treatment and half this without treatment, patients who were asymptomatic or who had a very poor prognosis in any case being excluded from the analysis. It is of interest that the survival with penicillin alone was rather less than with arsenic and bismuth either alone or combined with penicillin. Leonard and Smith point out that insufficient time has elapsed to permit a proper evaluation of penicillin therapy, but they suggest that in the interim it would perhaps be unwise to rely on it alone in the treatment of aortic incompetence.

Leonard and Smith's paper represents a great deal of patient and tedious work, and those who have occasion to assess the efficacy of penicillin therapy in the future will have cause to thank them for it. On the other hand, as the authors would agree (to judge by their examination of the earlier literature), their contribution must be read and interpreted with care.

JOSEF SKODA.

THE *Wiener klinische Wochenschrift* each year devotes one of its issues to an appreciation of the life and work of a famous physician or surgeon who helped materially to make Vienna renowned as a centre of research and teaching in medical science. Last year it was Josef (or Joseph) Skoda, who was chosen for a commemorative number, and the writing was entrusted to Erna Lesky, of Vienna.[1] It is curious that the two men who did most to give Vienna her renown in medical circles the world over, Skoda and Rokitansky, were heavily handicapped in boyhood by bitter poverty. Josef Skoda was born on December 10, 1805, the son of a struggling locksmith in Pilsen. He and his three brothers slept in an attic with roof so faulty that often in winter the boys would brush away the snow which had fallen on their beds. Their father could not afford candles or lamp; so the boys read school books and prepared their lessons by the glow of a stove with open door. Fortunately, the boy Josef attracted the attention of a woman silk manufacturer in Vienna, who gave him board, some teaching and five gulden a month in return for hard work. How he managed to matriculate and then pass through the medical course is a puzzle, but he did it. Physics and mathematics were his first loves, and the discipline of these studies influenced his subsequent medical investigations, in which he ever sought precision of diagnosis and description. His first medical job was to investigate the ætiology of cholera in Bohemia, and here he came to the conclusion that this disease was infectious, a view hotly contested by many in the profession. In 1833 he was appointed to an unpaid junior position in the famous Vienna hospital, and here he encountered conservatism and bumbledom in a high degree. He saved the life of a man by an emergency tracheotomy, but was severely reprimanded for not reporting the case before operation and not obtaining permission to conduct a tracheotomy. As a punishment he was compelled to spend three months as a junior physician in a lunatic asylum. Soon the irrepressible young doctor was experimenting with auscultation and percussion, improving the technique and clarifying the results. We today are apt to forget, if indeed we really know, how much we owe to Skoda in these two diagnostic procedures. Auenbrugger had introduced percussion in 1760, but his views regarding the origin of the sounds remained unchallenged until Skoda, helped by the brilliant pathological anatomy of his friend and colleague Rokitansky, put matters to rights. Auenbrugger and his successors believed that each organ emitted its own specific sound: there was a liver sound, a stomach sound, a heart sound, and so on. Skoda showed that, apart from the variations produced by alterations in the force of impact

[1] *Wien. klin. Wchnschr.*, September 28, 1956.

and those arising from subcutaneous adipose tissue and from bone, the air in and around each organ was largely responsible; and he drew attention to the changes in thoracic percussion caused by pleural effusion. Laennec invented the stethoscope in 1819, but it will hardly be believed today that until Skoda's investigations the place of a murmur in the heart cycle was disregarded.

Skoda as a junior physician scored more than one triumph in diagnosis. The French ambassador in Vienna, the Duc de Blacas, became seriously ill. Vienna's three leading physicians diagnosed an enfeebled liver and prescribed taking the waters at Karlsbad. Skoda proclaimed that the trouble was an aneurysm of the abdominal aorta and gave a grave prognosis, which turned out to be correct. A post-mortem examination proved that an aneurysm of the abdominal aorta was the cause of death. It is pleasant to record that one of the three doctors admitted the mistake and gave warm praise to the youthful researcher.

In 1839 there was published Skoda's great book on auscultation and percussion; and if that overworked adjective epoch-making should be employed at all, it certainly was applicable here. But jealousy of older doctors occasioned the simple-minded Skoda much unhappiness. Fortunately, French physicians were appreciative and responsive, and the word *scodisme* began to appear in French medical writings. An amusing result of this popularity outside his own country was that the big influx of foreign doctors into Vienna excited the suspicions of the police, who interrogated the visitors; when told that the visitors' desire to sit at the feet of Dr. Skoda was the cause of their invasion of Austrian territory, the police were surprised that a young doctor of whom they had never heard should win such renown abroad. Skoda's reforms affected treatment as well as diagnosis, and he may be considered largely responsible for the decline and ultimate disappearance of venesection, blisters, cupping, artificial ulcers, setons and the like. In 1841 he was promoted to be senior physician in the famous hospital, and in 1846 he became professor. He retired in 1871 and died in 1881.

One matter not mentioned in Lesky's biographical article is that there was probably some hereditary talent in the family, for Emil Ritter von Skoda, a nephew of the doctor, became a great industrialist, founding and owning the largest armament factory in Austria.

"FLUORTHANE"

ANY new compound which has anæsthetic properties is of great interest. The lodestone of anæsthesia is a compound with the potency and ease of administration of cyclopropane, which has the therapeutic safety of ether, and which is also non-explosive. This compound does not exist yet, but a group of compounds have been investigated within recent years which have the lodestone in mind. For possible use as inhalation anæsthetics, fluorine derivatives of hydrocarbons and ethers have been investigated. Some of them have in fact anæsthetic properties, but the anæsthetic effects are usually accompanied by intense hypotension and cardiac arrhythmias, particularly ventricular fibrillation. More recently tri-fluoro-ethyl-vinyl-ether has been investigated, but this is explosive in a concentration of more than 3% in oxygen. A later development has been the investigation of "Fluorthane". J. Raventos[1] has studied some of its physical properties and pharmacology. It is a volatile liquid with a specific gravity of 1·86 and a boiling point of 50·2° C. It is non-inflammable with oxygen between concentrations of 0·5% and 50%. It is stable over warm soda lime. It is an inhalation anæsthetic more potent than ether or chloroform, and with a therapeutic ratio twice that of ether. The only pathological lesion found, after prolonged exposure of animals to this substance, was some dilatation of the proximal tubules of the kidney, but without altered renal function; this is most probably a reversible reaction.

In Manchester, England, about 500 people received "Fluorthane" anæsthesia from Michael Johnstone.[1] The patients ranged from good to grave "risks", with the majority classed as intermediate. Using the "Trilene" bottle of a Boyle's machine, but with an extended scale, Johnstone obtained adequate vapour concentrations. He found that "Fluorthane" gave smooth and easily reversible rapid anæsthesia, with adequate relaxation. Although relaxation was of a high degree, there was no obvious respiratory depression. Hypotension and bradycardia were constant and roughly proportional to the concentration of "Fluorthane" vapour. This was consistent with an autonomic depression, and was usually accompanied by vasodilatation and sweating, whilst the usual respiratory secretions were absent. Vomiting was rare, and shock was not seen. These disadvantges of hypotension and bradycardia in Johnstone's cases were not severe; and the few cases of cardiac arrhythmia, none of which were fibrillation of the ventricle, were corrected with atropine. He found that the relaxant to be used with "Fluorthane" should be suxamethonium, as patients breathing "Fluorthane" were sensitive not only to curare but also to neostigmine. Indeed the dosage of suxamethonium should also be small. There were no deaths attributable to the new anæsthetic.

R. Bryce-Smith and H. D. O'Brien[2] in Oxford used "Fluorthane" either by the open drop method or through an E.M.O. inhaler. They also reported a smooth and rapid reversible anæsthesia, without the usual salivary and bronchial secretions. They found, however, in contradistinction to Johnstone, that depression of respiration was a disadvantage, and advise the limitation of use of the drug, at present, to skilled anæsthetists. However, they did note that the rapid, pleasant induction would be ideal for children.

These two reports on an anæsthetic of great promise are welcome; they may presage a useful future for "Fluorthane", since, if its hopes are realized, it would be an ideal anæsthetic in general practice. However, before that can be considered, much more work and many more papers are needed.

[1] *Brit. J. Pharmacol.*, December, 1956.

HEPATITIS FOLLOWING BLOOD TRANSFUSION.

IN one issue of the *Deutsche medizinische Wochenschrift*, namely, that of November 30, 1956, there are two articles devoted to the question of hepatitis following blood transfusion. One (page 1936) is a critical survey of the literature. It is astonishing, states the writer of the review, that the possibility of transmitting hepatitis by blood transfusion was not envisaged earlier. Apparently the incidence of such transmission in the second World War was very high, as was the mortality. There is a curious unanimity in the writings of many observers that transfusion with plasma was more apt to be followed by hepatitis than transfusion with whole blood, the cause of the difference being ascribed to the pooling of plasma, so that the virus of one donor could be spread over many recipients. Attempts have been made to sterilize plasma by ultra-violet irradiation, but these have not been satisfactory; a more promising procedure is to demineralize the plasma by ion interchange and then to apply pasteurizing heat, which in the absence of salts does not denature the proteins, though it destroys the virus.

The other article (page 1914) asks the question: How long after hepatitis can a donor's blood be used safely in transfusion? The two clinical researchers find that at least five years must elapse; this conclusion fits in with the views of other investigators of the same topic.

[1] *Brit. J. Anæsth.*, September, 1956.
[2] *Brit. M. J.*, October 27, 1956.

Abstracts from Medical Literature.

SURGERY.

Carcinoma of the Colon.

W. Suermondt and G. Korthof (*Arch. chir. neerl.*, Volume 8, Fasc. 2, 1956) describe a personal series of 170 patients suffering from carcinoma of the colon. They state that their observations run parallel with those of authors describing similar series of cases. Nevertheless, they describe a very interesting case of a patient who had simultaneous carcinoma of the caecum and tuberculosis of the caecum. This patient was a forty-year-old woman with a history of diarrhœa occurring in the morning for several years prior to treatment. Three weeks prior to admission to hospital she suffered pains in the right lower abdominal quadrant with nausea and a tendency to vomit. She had never noticed blood or mucus in the motions. Examination of the patient disclosed generalized abdominal distension and a hard tumour in the right iliac fossa. Operation disclosed a hard tumour in the caecum, to which the omentum adhered. As the caecum was being mobilized, a tuberculous abscess was opened. There were numerous large lymph nodes next to the caecum. Ileocolectomy was performed. Examination of the specimen showed an adenocarcinoma of the caecum, with metastases in the lymph nodes. There was also a caseating tuberculous mass of the caecum, which was involving the lymph nodes. The carcinoma recurred within a year of operation, and was completely inoperable.

Cancer in the Second Breast.

A. Kilgore, H. G. Bell and R. Ahlquist (*Am. J. Surg.*, August, 1956), in a series of 1200 radical mastectomies, found that cancers behaving like independent growths had developed in the second breast in 31 patients (2·6%). They consider that additional cases will yet appear among patients in the series who are still living. Of a group of stage I breast cancer patients, cancer developed in the second breast in 4%. The authors consider that if this figure is corrected for future cases among patients still living, the eventual incidence will apparently rise to about 6%, which is roughly three times the general incidence of unilateral cancer.

Post-Operative Pancreatitis.

E. Boles, junior (*Arch. Surg.*, October, (1956), states that post-operative pancreatitis ranks as a significant cause of death in upper abdominal surgery, particularly following operation on the biliary tract and stomach. He states that clinical recognition of the complication is often difficult, so that reliance on the serum amylase test in the early post-operative period is recommended if and when abdominal pain occurs. He considers that irrigation of the common bile duct in an effort to dislodge calculi is a reasonable mechanism of producing acute pancreatitis under anatomical circumstances which permit reflux of fluid up the main pancreatic duct under pressure. Severe pancreatitis may develop as a consequence of damage to the pancreatic duct when the duodenum is mobilized during the performance of a partial gastrectomy.

Surgical Management of the Complications of Reflux Œsophagitis.

F. Ellis, junior, H. Andersen and O. Clagett (*Arch. Surg.*, October, 1956) have reviewed a total of 45 patients with complications of reflux œsophagitis who have undergone operation at the Mayo Clinic in the previous ten years. The cause of this œsophagitis was œsophageal hiatal herniæ in 27 patients, excision or destruction of the cardiac sphincter in 10, and vomiting, either post-operative or secondary to obstructing duodenal ulcers, in six. The indications for operation were œsophageal obstruction in 20 patients, intractable pain in 17 and hæmorrhage in eight. Apart from repair of the herniæ they used a number of operative procedures, including œsophagogastrectomy of various types and subtotal gastric resection. Of the 41 patients who had been followed up, 22 only were considered to have good results. The authors were dissatisfied with the results of conventional surgical measures, and tried a new operative procedure which included excision of the lower part of the œsophagus and the cardia, bilateral vagotomy and excision of the lower part of the stomach, including the antrum. The proximal part of the stomach was anastomosed to the œsophagus and the distal end to the duodenum. The authors state that short-term results in the nine patients so treated were encouraging.

Richter's Hernia.

R. Gillespie, W. Glas, G. Mertz and M. Musselman (*Arch. Surg.*, October, 1956) have reviewed 20 cases of Richter's hernia. Nausea and vomiting accompanied by a palpable mass or localized tenderness over a potential hernial orifice were the commonest clinical findings. X-ray evidence of obstruction was found in less than one-half of the cases. The authors found that the mortality was highest when the continuity of the bowel was undisturbed, as the lesion was not recognized and operation was delayed. Treatment of this lesion consists of early operation and, if necessary, resection of the devitalized segment with end-to-end anastomosis of the bowel, and repair of the hernia.

Inguinal Hernia in Children.

C. Mueller and G. Rader (*Arch. Surg.*, October, 1956) recommend that exploration of both inguinal canals be performed in all children under the age of seven years who are treated for inguinal herniæ. The incidence of an unsuspected hernia or potential hernia is approximately 65% in the apparently unaffected groin.

Observations on Venous Thrombosis.

J. McLachlin and J. Paterson (*Arch. Surg.*, October, 1956) state that the common site of origin of venous thrombi is in the thigh and pelvic veins rather than in calf veins. They consider, therefore, that surgical ligation of the superficial femoral vein in pulmonary embolism, either prophylactic or therapeutic, does not appear to be basically sound. They also state that separation of thrombophlebitis and phlebothrombosis because of a supposed difference of embolic potential is not warranted. They consider that venous stasis in the veins of the pelvis and lower extremities, rather than changes in the underlying vein wall or abnormalities in the blood coagulability, is the important factor; and that the best prophylaxis against venous thrombosis is the maintenance of an efficient circulation in the deep veins of the lower extremities.

Preservation of Rectum in Familial Polyposis of the Colon and Rectum.

S. Coleman and C. Eckert (*Arch. Surg.*, October, 1956) discuss 13 patients with polyposis of the colon and rectum, all of whom were treated by endoscopic control of rectal polypi combined with total colectomy and ileo-proctostomy. In the reconstruction of the continuity of the bowel they used end-to-end anastomosis between the rectum and the ileum. They had no operative mortality, and the morbidity consisted of intraperitoneal abscesses in one case, and simple mechanical intestinal obstruction in two other cases. They found that bowel function was excellent in 12 patients, but only satisfactory in one patient owing to the occurrence of involuntary defæcation at night. Colectomy did not appear to retard growth or physical development when carried out in childhood.

Pseudocysts of the Pancreas.

R. Bradley and M. Klein (*Arch. Surg.*, October, 1956) state that pseudocysts of the pancreas are uncommon. They report five cases, two following acute alcoholic pancreatitis, one following acute pancreatitis with concurrent gall-bladder disease, one following chronic pancreatitis, and one following trauma to the pancreas. The authors state that external drainage of three of the cysts was a protracted procedure with drainage continuing from four to twelve months. The patients' chief complaint during this time was their inability to obtain employment while the drainage persisted, though otherwise they were symptom-free. Two patients required a secondary operation to remove the persistent sinus tract. The two patients with cysts drained internally had short and smooth convalescences. The authors state that the stomach is the logical place for drainage because of proximity and ease of anastomosis. Fears of food entering the cysts were unfounded because of rapid obliteration of cyst space and healing of the residual defect in approximately two weeks.

Treatment of Diverticulitis of the Colon.

H. Ransom (*Am. J. Surg.*, November, 1956) states that surgical treatment is indicated not only for the complications of diverticulitis of the colon, but also for chronic diverticulitis with recurring attacks. In such cases he recommends operation in order to prevent the grave complications of perforation, fistula, and obstruction. Definitive surgical treatment

should include resection of the diseased segment of the bowel with ultimate restoration of continuity. Colostomy is almost never necessary. A three-stage operation, consisting of right transverse colostomy, resection, and closure of the colostomy, is the safest procedure. It is indicated in the case of perforation with pelvic abscess, or general peritonitis, in patients with obstruction, and in patients with fistula of the bladder or other sites. The one-stage operation may be safely employed for patients with recurring episodes of diverticulitis, for those with hæmorrhage, and in those cases in which carcinoma is suspected or cannot be excluded. In a series of patients treated at the University of Michigan Hospital hæmorrhage was not an important complication, being noted in only 17% of the cases. The author states that when hæmorrhage is the chief or sole symptom, and when it occurs frequently, carcinoma should be suspected and operation should not be delayed. In his group of cases fistula of the bladder did not present any particular technical problems. When a three-stage operation was performed, the fistula had completely or practically closed by the time of the resection.

Bleeding Œsophageal Varices.

W. McDERMOTT, JUNIOR, J. WAREHAM AND A. RIDDELL (*Ann. Surg.*, September, 1956) report a series of experimental and clinical studies designed to clarify the roll of massive gastro-intestinal hæmorrhage in precipitating so-called "hepatic coma". As a result of this, they present evidence to show that, in the presence of spontaneous or surgically constructed portal-systemic shunts, the introduction of blood into the gastro-intestinal tract is accompanied by striking rises in the level of ammonia in the peripheral blood. They state that if the rises in blood ammonia content are sufficiently high, central nervous system symptoms appear, ranging from mild confusion and disorientation to deep coma. Frequently these symptoms are reversible, but the clinical syndrome may terminate fatally. The rationale of these observations is fairly clear. Both in the dog and in man it has been shown that the portal vein consistently contains a large amount of ammonia nitrogen, which fluctuates with the amount of nitrogenous material taken into the gastro-intestinal tract. From the available evidence it appears that this ammonia nitrogen is derived from the action of amino acid oxidase and urease on ingested protein and other nitrogenous substances. These enzymes are apparently derived from the intestinal microorganisms. Normally, this ammonia is synthesized to urea in the liver, which thus forms a protective barrier against the accumulation of toxic levels of ammonia in the systemic circulation. In the presence of spontaneous or surgically constructed portal-systemic shunts, however, channels exist whereby this ammonia derived from enzymatic breakdown of nitrogenous material can bypass the liver and cause abnormal rises in the level of ammonia nitrogen in the peripheral blood. It is apparent from these observations that the introduction of blood into the gastro-intestinal tract in the presence of portal-systemic shunts may cause both hyperammoniæmia and central nervous system symptoms. The authors state that the use of L-glutamic acid will bring about a reduction in raised levels of blood ammonia and is a valuable adjunct in the treatment of ammonia intoxication (so-called exogenous hepatic encephalopathy). They consider that these studies form the background for a suggested programme in the management of bleeding œsophageal varices, with the following features: (i) Control of hæmorrhage—hæmorrhage must be controlled even though there is no apparent threat of exsanguination, as it is imperative to prevent the accumulation of a nitrogenous pool in the gastro-intestinal tract; this may be accomplished initially by balloon tamponade and even by direct transthoracic ligation of the varices. (ii) Elimination of the source of absorbed ammonia by purgation and enemata to remove the blood from the gastro-intestinal tract. (iii) Antibiotics—by depressing the bacterial count in the gastro-intestinal tract, the sources of urease and aminoacid oxidase can be controlled. (iv) L-glutamic acid—this is a valuable adjunct to therapy and may be given orally or parenterally in the glutamate form as individual circumstances demand.

Rubber Tubes and Anastomoses of the Common Bile Duct.

W. SILEN, D. L. MAWDSLEY, W. L. WEIRICH AND H. J. McCORKLE (*Arch. Surg.*, June, 1956) became interested in the subject of the relationship of a rubber tube with recently constructed anastomoses of the common bile duct, and conducted a study using 25 dogs. They set out to determine whether prolonged contact of a recently constructed anastomosis of the common bile duct with a rubber tube was associated with a greater incidence of stricture formation, and, secondly, to establish, if possible, the optimum period of time for splinting of the duct following anastomosis. Two operations, one week apart, were performed in 25 dogs. The first operation consisted of cholecystectomy and occlusion of the distal end of the common bile duct immediately above its entrance into the duodenum; at the second operation, the common bile duct was transected two to three centimetres above the occlusion and the cut ends were approximated in an end-to-end anastomosis. In one-half of the animals a choledochotomy was made two centimetres above the anastomosis and a "T" tube of rubber was placed in the ducts with one of the short limbs extending across the anastomosis. In the other half of the animals the "T" tube was so placed that the lower limb was at least one centimetre above the anastomosis. The "T" tubes were allowed to remain in place for various periods of time after the anastomosis—that is, three weeks, three months and six months. The animals were then sacrificed, and detailed examination was made at autopsy. Two of the dogs died before the completion of the experiment, and of the 23 dogs studied none developed strictures at the site of ligation of the common duct. One only developed a very slight narrowing of the suture line. Calculi were found in the extrahepatic biliary system of two dogs in which the "T" tubes had remained in place for six months. After surgical division and anastomosis healing was complete in all groups of experimental animals. Thus, prolonged contact of an anastomosis of the common bile duct with a rubber tube does not appear to be associated with an increase in the inflammatory reaction in and about the anastomosis, or with an increase in the incidence of stricture of the duct.

Treatment of Meconium Ileus by Ileo-transverse Colostomy.

T. D. THROCKMORTON AND P. M. PORTER (*Arch. Surg.*, June, 1956) report cures in two patients with meconium ileus by ileo-colostomy and immediate decompression with a proximal Witzel ileostomy. Meconium ileus is the earliest manifestation, and the most acutely grave complication of a generalized disease, fibrocystic disease of the pancreas, mucoviscidosis or "eccrinosis". The diagnosis and treatment of this condition are a matter of considerable urgency and cannot await some of the niceties now available for confirmation. The authors report the many attempts at the surgical cure of meconium ileus with the results obtained. They summarize the work of Sivenson, Hiatt and Wilson, Gross and others. They state that Gross now favours Mikulicz resection and double ileostomy, and since 1949 has had a 79% survival rate. In the two cases reported, the authors chose to do primary side-to-side ileo-transverse colostomy with complementary Witzel ileostomy because of certain advantages: (i) It is a single-stage procedure. (ii) Immediate restoration f intestinal continuity is afforded. (iii) Immediate decompression of obstructed intestine is permitted. (iv) A means of irrigation is provided by which pancreatic enzymes may be used to liquefy abnormal meconium. (v) No intestinal resection is done, nor does one seem warranted, considering that there exists no lesion intrinsic to the intestinal wall. (vi) There is reason to believe that the terminal part of the ileum and right side of the colon do not remain defunctionalized. (vii) The disadvantages of a double-barrelled skin ileostomy, no matter how brief, and how minimized, are averted.

The Closing Mechanism of the Cardia.

J. NAUTA (*Arch. Chir. Neerl.*, 1956) studied the closing mechanism between the œsophagus and stomach in dogs with the aid of radiocinematography, endoscopy and anatomical examination. The œsophageal wall itself possesses no demonstrable mechanism of preventing regurgitation. The closure between the œsophagus and the stomach is valve-like in nature owing to the oblique inlet of the œsophagus into the stomach. The outside of this oblique inlet is supported by the diaphragm, while the inside is supported by the oblique fibres of the stomach. The oblique inlet is therefore an important factor in the satisfactory closure between stomach and œsophagus. A rosette of gastric mucosa at this site is essential for water-tight closure, and the rosette itself is supported by the diaphragm and the oblique fibres.

British Medical Association News.

NEW SOUTH WALES FEDERATION OF COUNTRY LOCAL ASSOCIATIONS.

Fifth Annual Post-Graduate Course.

The fifth annual post-graduate course of the New South Wales Federation of Country Local Associations (FOCLA) was held at Lismore from August 13 to 17, 1956.

Edward Albert Woodward.

Dr. R. E. Longworth (Lismore) delivered an oration in memory of the late Dr. E. A. Woodward (see page 301).

Gastric Ulcer.

Dr. T. M. Greenaway (Sydney) read a paper entitled "Gastric Ulcer: Some Interesting Aspects" (see page 304).

Dr. Evan Thomson (Brisbane) read a paper entitled "Indications for Surgery in Peptic Ulcer" (see page 306).

Dr. B. Cook (Bulli), from the chair, thanked the speakers for their informative addresses, and asked about the place of ganglion-blocking agents in treatment.

Dr. Greenaway replied that there was some evidence that none of those agents had any influence at all.

Dr. N. E. Brand (Lismore) thanked Dr. Thomson for pointing out that there was a definite place for gastrectomy in the treatment of gastric ulcer.

Dr. E. M. Kent Hughes (Armidale) asked Dr. Greenaway if he really advised that patients might almost immediately have food, even corned beef and indifferently cooked cabbage, and also whether they might smoke numerous cigarettes.

Dr. Greenaway was convinced that clinicians flattered their own egos by learning a routine and selling it both to themselves and to their patients. By sticking strictly to so-called "ulcer diets" they were inclined to reduce the patient to a low nutritional status. What was needed was contentment as well, and that was more easily obtained by full diet and rest.

Dr. J. V. Champain (Tenterfield) asked about the relationship between gall-stones and peptic ulcer.

Dr. Thomson considered that there was no direct ætiological connexion between the two conditions; they were both very common, and must inevitably be seen combined in the same patient. In view of the diagnostic similarities, he thought that it was often wise to have a barium meal X-ray examination performed as well as cholecystography.

Dr. K. G. Lawrence (Lismore) asked Dr. Greenaway whether, when a patient presented with an ulcer, he immediately ordered bed rest, or might try two or three weeks' ambulatory treatment at first.

Dr. Greenaway replied that that was an important question economically, and on occasions it might be necessary to use some compromise.

Dr. D. B. Sillar (Lismore) inquired about the place of gastro-enterostomy in treatment, as in the case of outlet obstruction.

Dr. Thomson, in reply, said he thought that gastro-enterostomy still had a definite place in the surgery of peptic ulcer—not in gastric ulcer, but in many cases of duodenal ulcer. It was the operation of choice for female patients and for older men. Sir Gordon Gordon-Taylor had expressed the view that it was a surgical crime to perform a gastrectomy for duodenal ulcer in women. That was important in connexion with the absorption of iron from the duodenum, for which hydrochloric acid was necessary, and gastrectomy both removed the acid and put the duodenum out of action.

Dr. G. J. Rawle (Lismore) referred to the good results of vagotomy, thoroughly performed, in some cases of gastric ulcer, to the facts that fatty foods reduced the formation of gastrin, and hence the quantity and quality of gastric juice, and that a high glucose level in the blood tended to inhibit the secretion of hydrochloric acid. He asked whether, in view of those facts, any work had been done on dietary for gastric ulcer to implement the idea that a high fat intake and a full diet would otherwise help in the treatment of gastric ulcer.

Dr. Greenaway, in reply, said that much experimental work had been done along those lines. It had to be remembered that gastric ulcer was different from duodenal ulcer. In gastric ulcer there need not be the same degree of hyperacidity and hypersecretion, and it had been recorded with achlorhydria. The secretion was actually lower in gastric ulcer than in duodenal ulcer. Also the question of psychosomatic treatment had to be considered.

Dr. Thomson reminded listeners that gastric ulcer might occur after vagotomy.

Dr. B. A. Frith (Lismore) asked about the question of operation in intractable cases when the patient had had adequate rest and medical treatment, or in recurrences in patients insufficiently treated.

Dr. Thomson said that he himself always treated patients medically at first.

Dr. N. Sullivan (Dubbo) asked whether it was a fact that duodenal ulcer was more subject to psychological influences than gastric ulcer.

Dr. Greenaway replied that he was in favour of surgical treatment in any case of intractable ulcer, and in cases of hæmorrhage.

Dr. L. Roberts (Lismore) asked whether it was still held, as stated by Hirst nearly thirty years earlier, that hydrochloric acid was the factor which prevented peptic ulcers from healing.

Dr. Greenaway replied that he believed that antacid therapy helped healing.

Dr. N. J. Rogers (Lismore) asked whether the secondary rise in gastric acidity influenced the suggested treatment following the evening meal, either with an antacid only, or with supper as usual.

Dr. Greenaway, in reply, said that the nocturnal rise occurred whether food had been taken or not. He believed in giving supper, as well as a long-acting antacid with an adsorbent action rather than a neutralizing action.

Dr. Rogers further asked what influence the presence of gastric ulceration had on anticipated cortisone therapy.

Dr. Greenaway replied that most of the corticoids were ulcerogenic, but that the newer prednisolone was more antipyretic and was preferred. However, it would be better not to give any corticoids in the presence of an ulcer.

Disordered Uterine Action.

Professor G. Shedden Adam (Brisbane) read a paper entitled "Disordered Uterine Action".

Hazards of Multiparity.

Professor G. Shedden Adam (Brisbane) read a paper entitled "Some Hazards of Multiparity" (see page 309).

Dr. T. Roberts (Lismore) asked first about the hazards of the development of Rh agglutinins in *multiparæ*. It had been said that they could appear at three periods of pregnancy—namely, at ten weeks, at thirty-two weeks and at term.

Professor Adam agreed that the blood should be tested at those intervals, but thought that one should not stop at thirty-two weeks if there was reason to believe that agglutinins could develop later. He thought that to do it properly, one should test the blood at thirty-six weeks and again at term. The most difficult problem was that of the young *primipara* who forgot or did not know about a previous blood transfusion.

Dr. Roberts asked further for Professor Adam's views on the management of post-partum hæmorrhage, and whether he recommended the intravenous use of ergometrine.

Professor Adam said that the use of ergometrine before the placenta was out was a new development, and it was now thought that it was a useful compromise with certain indications. Those indications included established hæmorrhage within a few minutes of the birth, a history of previous manual removal of the placenta, a history of previous antepartum hæmorrhage, and a history of over-distended uterus. He preferred to give 0·5 milligramme rather than 0·125 milligramme, and reminded listeners that ergometrine could be given intramuscularly if the intravenous route was not convenient.

Dr. Roberts finally asked about the management of anæmia in a *multipara*.

Professor Adam said that ferrous gluconate should be given for the last three months of pregnancy in subtropical climates. He mentioned the investigation recently under-

taken in Brisbane, in which a group of patients at an antenatal clinic had been compared with hospital nurses. A 10% difference had been found, the public hospital patients averaging 70% hæmoglobin value.

DR. E. KENT HUGHES asked Professor Adam for his views on the modern trend towards getting patients out of bed early after confinements, in two to four days.

Professor Adam said that he was in no doubt that patients should not be got out of bed and sent home early. It was better to keep the patients in hospital for a full seven days.

DR. F. NICHOLL (Lismore) asked about the risks of labour induced at eight months in cases of previous Cæsarean section or disproportion.

Professor Adam said that there were two essentials to consider: (i) whether the previous indication for Cæsarean section no longer existed, and (ii) whether everything was normal. He was doubtful whether induction of labour should be performed in such cases, but the patient in any case should certainly not be given "Pitocin".

Psychosomatic Diseases in Children.

DR. FELIX ARDEN (Brisbane) read a paper entitled "Psychosomatic Diseases in Children" (see page 312).

DR. T. M. CLOUSTON (Sydney) opened the discussion by congratulating Dr. Arden on his human and humane approach to a difficult subject. He said that one of the difficulties lay in defining what was meant by psychosomatic medicine. With regard to lonely children, he thought that if such a child had to go to hospital, it would be difficult to induce him to leave his mother again. A similar problem occurred in the case of a mother returning from hospital with a baby. With regard to thumb-sucking, Dr. Clouston said he thought that if a baby was allowed a dummy, he nearly always spat it out at between seven and nine months, and never indulged in thumb-sucking. Dr. Clouston commended Dr Arden's remarks about consultation between the school-teacher and the mother who knew about the problem. With reference to food refusal, Dr. Clouston mentioned an investigation at two orphanages in the United States of America, in which carefully weighed diets were given at one, while at the other the children were given unrestricted diets. It had been found after six months that the children who were fed completely freely did better than those on a diet, and ate all the essential foods.

Dr. Arden agreed with Dr. Clouston's remarks about the lonely child, but mentioned in that connexion that more and more hospitals were allowing unrestricted visiting of children. There was another scheme being tried whereby children were kept out of hospital as much as possible, and nurses, physiotherapists and the like were sent out to them. Dr. Arden agreed about the harmlessness of dummies.

DR. E. KENT HUGHES (Armidale) agreed about the use of dummies for babies as a preventive of thumb-sucking, but thought that it became a more difficult problem in older children, whether with the thumb or two fingers. Concerning food problems, she thought that the best attack on a child refusing food was to send him to bed for a few hours. Dr. Kent Hughes mentioned the problem of the baby health centre with an over-anxious and conscientious sister transmitting her anxiety to the mothers. Another problem which concerned Dr. Kent Hughes was the anxiety in older children about examination results and competition. She thought that it was better to have no actual competition, with no marks or places in class, and that something should be done about minimizing the strain on children produced by early examination competition.

Dr. Arden thought that the thumb-sucking older child was usually an introverted only child with nothing to do but occupy himself with his own body. He agreed with what had been said about the nurses at health centres, but thought that that problem mainly concerned the older nurses. The best slogan for mothers was "enjoy your babies", and they should not worry about them. With regard to school stresses, Dr. Arden thought that the individual approach by an individual parent to the schoolmaster was no good. Sometimes an approach by the doctor could be useful. The main difficulty was with trying to bring the slightly under-average child up to average.

DR. ADRIAN PAUL (Gosford) pointed out the problem of the bullying teacher, and the danger of his being interviewed by the parent, particularly by the angry parent. He also thought the problem of books for parents to read was a difficult one.

Dr. Arden agreed with Dr. Paul's remarks about the angry parent.

DR. J. G. OPIE (Lismore) recounted a bad experience at the age of five years involving an anæsthetic for tonsillectomy. He thought that the best way out of that problem was by adequate sedation, especially by the rectal administration of thiopentone if necessary. In any case, he thought that a child should always be thoroughly sedated before any such experience.

Dr. Arden supported that view, and thought that mental trauma was often administered by the nurse unnecessarily, as by giving penicillin injections.

DR. R. E. LONGWORTH (Lismore) agreed about the value of sedatives before anæsthesia.

DR. A. J. OPIE (Lismore) did not agree with Dr. Kent Hughes that competition should be abolished, as he did not like the idea that people should have to dodge everything. He stressed the problem of abdominal pain in childhood. He always thought that such a child might have some organic disorder in its abdomen, and the pain should not always be regarded as psychosomatic.

DR. H. TAIT (Brisbane) thought that the flight from a problem into hysterical pain was a sign of poor adjustment.

Hypertension.

A symposium was held on the subject of hypertension; the medical aspects were dealt with by Dr. T. M. Greenaway (Sydney), the obstetrical aspects by Professor G. Shedden Adam (Brisbane), and the surgical aspects by Dr. Evan Thomson (Brisbane).

PROFESSOR G. SHEDDEN ADAM (Brisbane) said that he would deal almost entirely with essential hypertension, or hypertensive vascular disease. It had a relatively high incidence, and its sequelæ could be serious. The still-birth rate among infants of hypertensive mothers was approximately six times the over-all rate. As recently as 1947, an overseas investigation of 300 hypertensive pregnancies had revealed a maternal death rate of 2%, compared with an over-all maternal death rate of about one per 1000 births. Professor Shedden Adam mentioned the risks to both mother and fœtus which essential hypertension could produce, and then discussed the diagnosis. He said that a blood pressure raised to 140 millimetres of mercury, systolic, and 90 millimetres, diastolic, early in pregnancy, was probably due to essential hypertension. However, it was impossible to be sure that the condition was not preeclampsia unless the patient's blood pressure before pregnancy was known. It was unwise to take an arbitrary time, such as the twentieth week of pregnancy, to make the final diagnosis; it could be made only by regular observations of the patients *post partum*. In exceptional cases the physical signs of long-standing essential hypertension might be found, but in the majority they were absent. Nevertheless, a reasonably accurate presumptive diagnosis of essential hypertension could usually be made. The clinical course of a pregnancy in a woman already suffering from essential hypertension would be a worsening of the blood pressure readings in rather more than half the cases encountered. That was probably the result of placental ischæmia, which inactivated at least one enzyme normally antagonistic to vasopressor influences. Whilst only a few mothers would come to any harm as a result of this, the consequences, both immediate and remote, of vascular catastrophes, whether cerebral or intrauterine, were likely to be serious. Pregnancy toxæmia was at least twice as common in hypertensive as in normotensive pregnant women, and might then take a more severe form. Fœtal wastage tended to be high, and treatment was only partially effective. If the hypertension was further complicated by preeclampsia, 46% of patients would suffer a permanent rise of blood pressure of more than 20 millimetres of mercury; the corresponding figure in the absence of toxæmia was 30%. Advice was often asked as to whether a hypertensive woman who had had a "hypertensive pregnancy", perhaps ending in loss of the fœtus, should undertake another pregnancy. The risks were greatest with young *primiparæ*, since hypertension was a more serious condition in young patients and toxæmia was more likely in *primiparæ*. A second or third well-supervised pregnancy would often proceed uneventfully; but increasing age and parity brought risks of hypertensive cardiac failure or even of renal insufficiency and failure. The risk of cerebral vascular accident did not seem to be related to parity.

Discussing treatment, Professor Shedden Adam said that in the rare instances of malignant hypertension early in pregnancy, the pregnancy should be terminated. In other cases of hypertension in pregnancy, the pregnancy should be allowed to continue. The most important single step in treatment was rest in bed, either at home or in hospital according to the relative rise in the diastolic blood pressure. Admission to hospital was urgently necessary if even slight

signs of toxæmia developed. After a pregnant hypertensive patient had been admitted to hospital, essential hypertension was treated by the usual measures. Associated preeclampsia was treated in the same way as in the normotensive patient, and if it was not severe it would usually respond satisfactorily. Professor Shedden Adam said that, apart from the reserpine compounds, he had made little routine use of powerful hypotensive drugs. He discussed the possible indications for the use of such drugs, and his reasons for not using them. He said that, although he did not use hypotensive drugs, he was far from satisfied with the existing methods of treating hypertension complicating pregnancy. The whole position of drug therapy for that condition should be regarded as fluid. In the meantime, the use of barbiturates and tranquillizing drugs of the reserpine group and the occasional use of veratrum compounds, combined with bed rest and well-timed induction of labour, would minimize the risk of serious maternal complications and safeguard the fœtus as much as possible in the light of present knowledge. Professor Shedden Adam then referred to the case in which toxæmia developed in a hypertensive patient as early as the twenty-eighth to thirtieth week of pregnancy. He discussed the risks to the fœtus and to the mother if the pregnancy was (*a*) terminated then or (*b*) carried on to the thirty-fourth or thirty-fifth week, labour being then induced by artificial rupture of the membranes. He thought that, if the physical signs did not become progressively worse, the latter course was preferable. However, even in the absence of albuminuria, a persistently raised systolic blood pressure of over 200 millimetres of mercury, or a diastolic pressure rising in spite of treatment, was an indication for induction of labour in the presence of a viable infant. With lesser degrees of hypertension, it was good practice to terminate the pregnancy no later than the thirty-eighth week. In severe cases when premature delivery was indicated, particularly in *primiparæ*, it was often carried out by Cæsarean section; but fœtal survival rates could be disappointing. Induction of labour by rupture of the membranes was often surprisingly successful in such cases; episiotomy or gentle forceps delivery reduced the risk of intracranial hæmorrhage in such premature infants. When rupture of the membranes failed to induce labour within a reasonable time, Cæsarean section could still be undertaken without much risk of infection. Evacuation of the uterus of hypertensive patients by either method was often followed by a considerable fall in both systolic and diastolic blood pressures within a few hours, followed by a rise over the next few days. During the period of fall the patient's condition might appear to have deteriorated, and signs resembling those of shock might be present. In such cases it might be wise to control the rate and extent of the fall by the intravenous drip administration of glucose solution, the rate of flow being governed by the blood pressure readings.

DR. EVAN THOMSON (Brisbane) said that the surgeon was interested in hypertension for two reasons; first, he had to operate on hypertensive subjects for various surgical disorders, and secondly, surgery had a place in the management of a number of conditions in which hypertension was either the sole or a major feature. Referring first to surgery in hypertension, Dr. Thomson said that hypertension itself did not greatly increase the risk of operation provided that renal function was adequate and that no cardiac complications were present. However, there was a slight but real risk of post-operative cerebral vascular accidents in hypertensive patients. Cardiac failure was unlikely unless there had been previous episodes, and both pulmonary infection and pulmonary embolism were more common in patients with cardiac damage than in others. Hypertensive patients ran a real risk of post-operative coronary occlusion, especially if they had coronary disease, and no patient who had had *angina pectoris* or a coronary occlusion was a favourable "risk". Renal failure might follow operation, and even when renal function was good there was a risk of uræmia. The precipitating factor in all the important complications was prolonged hypotension; a stable blood pressure must be maintained during operation, and post-operative shock must be avoided.

Discussing the surgery of hypertension, Dr. Thomson referred to Pickering's classification. The first group included conditions characterized by systolic hypertension due either to increased cardiac stroke output or to increased aortic rigidity. Among such conditions were patent *ductus arteriosus*, arterio-venous fistulæ (congenital and post-traumatic), Paget's disease of bone, and thyrotoxicosis. Pickering's second group comprised cases of hypertension in which both systolic and diastolic pressures were raised; this group was divided into two subgroups, secondary hypertension and essential hypertension. Among the prominent surgical causes of secondary hypertension were diseases of the urinary tract. Hypertension might respond to relief of the obstruction caused by prostatic enlargement or by calculi. Hypertension might be due to unilateral kidney disease (renal tumonrs, vascular lesions such as renal artery compression and aneurysm, hydronephrosis and tuberculosis of the kidney); but examples of the relief of hypertension by nephrectomy were not common. The most important unilateral renal lesion with hypertension was chronic pyelonephritis, particularly in a small, hypoplastic kidney, and some improvement might follow removal of such a kidney; care should be taken to see that the other kidney was normal, and in general the operation should be performed for the renal abnormality rather than for the hypertension. Thoraco-lumbar sympathectomy should possibly be combined with nephrectomy. Coarctation of the aorta would be missed if the femoral arteries of all hypertensive patients were not examined as a routine measure; it should be recognized and surgically treated by the age of ten or twelve years, if its bad results were to be prevented.

Dr. Thomson then referred to the endocrine tumours, particularly phæochromocytomata. He said that although such tumours were most commonly adrenal, they might arise anywhere in the sympathetic system. Adrenal tumours involved the right side more commonly than the left, and 10% were bilateral. They produced hypertension by the release of large amounts of nor-adrenaline and adrenaline, the former predominating. Patients might present with paroxysms of hypertension, or with persistently raised blood pressure. The history might extend over many years. The most useful diagnostic test was the estimation of catechol amines in the urine; plain radiography, excretion urography and perirenal insufflation of air might aid in localization of the tumour. Diagnostic laparotomy might be required. Operation was necessary for cure, but was a hazardous procedure; a fatal paroxysm of hypertension might be produced by handling of the tumour, and "Regitine" could be administered before the tumour was touched, in order to obviate that occurrence. The blood pressure suddenly fell after removal of the tumour, and gross and fatal hypotension might ensue; at that stage 1-nor-adrenaline should be administered by the intravenous drip method. Dr. Thomson then spoke of Cushing's syndrome. He said that hypertension was usual in association with it, and it appeared certain that the various features of the condition were the result of the influence of hormones produced in the adrenal cortex. Although the known hormone disturbances did not appear to explain the hypertension, the removal of an adrenal tumour, or total or subtotal resection of both adrenals in the absence of a tumour, reduced the blood pressure in most cases of Cushing's syndrome. Referring to essential hypertension, Dr. Thomson said that surgery played a definite but small part in its management. Four main types of sympathectomy had been used, and the operation of bilateral total adrenalectomy had now been added. The aims of the operations had been: (i) to reduce sympathetic tone and lower basal blood pressure levels; (ii) to modify the reflex vasoconstrictor control of blood pressure and thus abolish sudden elevations; (iii) to reduce reflex secretion of adrenaline; (iv) to stabilize blood flow through denervated viscera. The results might be good, though not necessarily permanent. It was fair to say that operation was indicated in cases in which medical treatment had failed. In many circumstances adequate control of drug therapy was impossible, and sympathectomy should then be seriously considered. In the opinion of Sir Horace Evans, the best results from operation were obtained in three groups of patients: (i) in young patients with severe hypertensive disease and pronounced retinitis, especially young women in whom the hypertension had first been noticed during a pregnancy; (ii) in patients suffering impaired vision from hypertensive retinitis, without severe cardiac or renal failure; (iii) in patients with severe hypertensive symptoms, particularly headache. Dr. Thomson said that operation should not be performed on patients aged over fifty years, or in the absence of symptoms. Congestive cardiac failure was a contraindication, but cerebral vascular accidents with complete recovery were not. Smithwick's operation should not be performed on patients with coronary artery disease. Total thoracic sympathectomy would relieve anginal pain, but frequently left prolonged and total disability. Among the undesirable side effects of operation Dr. Thomson mentioned the following: intractable intercostal neuralgia; postural hypotension; loss of sweating in denervated areas of skin, with excessive perspiration in the remaining skin. The performance of the Smithwick operation on both sides resulted in loss of the power of ejaculation in the male; power of erection might be reduced, but impotence was very uncommon.

Rheumatic Fever.

A symposium was held on the subject of rheumatic fever. Papers were read by Dr. Felix Arden (Brisbane), Sir Alexander Murphy (Brisbane) and Dr. T. M. Greenaway (Sydney).

DR. FELIX ARDEN (Brisbane) said that his part in the symposium was to discuss the often difficult matter of the diagnosis of rheumatic fever and to say a little about the various modes of onset seen in children. Perhaps that could best be done by referring to the report of the Rheumatic Fever Working Party of the Medical Research Council of the Privy Council in Great Britain and the Council on Rheumatic Fever and Congenital Heart Disease of the American Heart Association. Those two bodies, beginning in 1950 a joint controlled study of the efficacy of certain drugs in treatment, were at once faced with the necessity of standardizing their diagnostic criteria. That had to be an arbitrary business, as they could find no single symptom or sign common to all cases of the disease. A diagnostic scheme was finally agreed upon, the headings of which had been reproduced; Dr. Arden said that he proposed to amplify it in the wording of their report, and then to comment briefly upon the conditions listed. There were five major manifestations: carditis, polyarthritis, choreiform movements, subcutaneous nodules and *erythema marginatum*. Carditis had to be shown by any one of the following: (*a*) the development of an organic, apical, systolic murmur or an aortic diastolic murmur; (*b*) a change in heart size of more than 15% on standard X-ray film by any standard method of measurement; (*c*) pericarditis revealed by friction rub or effusion; (*d*) congestive failure in the absence of other causes, as shown by dyspnœa, orthopnœa, liver enlargement, basal pulmonary râles, increased jugular venous pressure or œdema. Polyarthritis was defined as pain and either limitation of active movement or tenderness in two or more joints. Movements of chorea had to be of at least moderate severity. There were also five minor manifestations—fever, elevated sedimentation rate, evidence of preceding streptococcal infection, increased *P-R* interval and evidence of a previous attack. Fever was defined as elevation of the temperature above 99·3° F. at least twice in any one period of twenty-four hours, or above 100·3° F. on any one occasion. Elevated sedimentation rate meant a rate of 15 millimetres or more in an hour on the Wintrobe scale, corrected to a hæmatocrit of 45%. Evidence of preceding streptococcal infection was determined by (*a*) culture of a throat swab in which β-hæmolytic streptococci predominated, (*b*) an antistreptolysin O titre of 200 units or greater, or (*c*) a reliable history of sore throat with fever, preceding the onset of the illness by an interval of one to four weeks. An increased *P-R* interval was defined as a value of at least 0·04 second beyond those given in standard tables for ages under sixteen years. Evidence of a previous attack was shown by a reliable previous history of rheumatic fever as defined in the criteria stated, or by evidence of preexisting rheumatic heart disease—namely, an organic apical systolic bruit or an apical diastolic or basal diastolic murmur.

Dr. Arden went on to say that, given the foregoing major and minor manifestations, the making of a diagnosis of rheumatic fever sounded rather like bidding on a contract bridge hand. The occurrence at one time of at least two major manifestations, or of one major and two minor manifestations, sufficed. But even with that ready-made scheme there were difficulties and uncertainties, because it was never possible to consider sick people in terms of simple arithmetic. He would therefore go over some of the symptoms and signs again. Referring first to carditis, Dr. Arden said that it was important to recognize and exclude the functional murmurs—soft, basal systolic murmurs, sometimes musical or squeaky, never blowing, harsh or heard at the apex. Trouble might be experienced with children with congenital heart lesions coupled perhaps with joint pains or growing pains. The resemblance to rheumatic fever might be very close, particularly if the condition of the heart was not known previously. A few points were helpful. One was that very few congenital heart murmurs exactly mimicked the mitral regurgitant murmur of rheumatic fever, either in the quality of the sound or in the site of maximum intensity and direction of conduction. Congenital lesions of the mitral valve itself were very uncommon. Another point was that it would be most unlikely for a child to develop rheumatic carditis of sufficient degree to produce a murmur, without at the same time having a significant degree of tachycardia, electrocardiographic changes or increased sedimentation rate. Development of an aortic diastolic bruit without signs of mitral involvement was unusual, but was very properly allowed for in the diagnostic scheme. It had happened in 1956 at the Brisbane Children's Hospital in a child who had been admitted with chorea, and it was the first manifestation of carditis. Pericarditis also might be an initial symptom and the cause of serious diagnostic error. A child had been sent to hospital in 1955 with the diagnosis of acute appendicitis, suffering from severe upper abdominal pain with fever and abdominal rigidity. A high appendix was suspected, but the surgeon stayed his hand, not feeling quite certain. Next day pericardial friction was evident. Dr. Arden said that he had seen another child on whom, under similar circumstances, laparotomy had been performed. The final diagnostic provision under "carditis" allowed for those children who presented with congestive heart failure. That was also an uncommon event, but was most likely to happen in young children in whom the tell-tale joint symptoms might be entirely absent, and whose only complaint might be one of listlessness and fatigue. Once again, under such circumstances it might be difficult to exclude congenital heart disease with subsequent failure. Patients with atrial septal defect were most likely to behave in that way. The only distinguishing feature might be the position of the systolic murmur—at the apex in rheumatic carditis, in the second and third intercostal spaces close to the sternum in atrial septal defect.

Turning to polyarthritis, Dr. Arden said that it should be noted that the committee insisted on two or more joints being involved, thus excluding the great majority of children with osteomyelitis, synovitis of the hip or suppurative arthritis. However, rheumatic arthritis could, and usually did, begin with one joint only, and the patient might be brought for treatment before others had had time to become involved. The appearance of the joints was helpful. The swelling was not great as a rule, and free fluid could hardly ever be detected. In an acutely involved joint any passive movement was extremely painful; but while there might be generalized tenderness, there was no one point of acute sensitivity over a bone. With osteomyelitis the reverse was the case. With care one particular bone, and one point on that bone, was extremely sore to touch, whereas if gentleness was exercised a fair range of passive movement could be obtained. As had been mentioned before, the smaller the child, the less pronounced were the joint symptoms likely to be. A characteristic of the rheumatic joint, regular enough to be included in any diagnostic scheme, was the quick response to salicylate. Only occasionally had he known that to be unreliable.

On the subject of chorea, Dr. Arden joined issue with the committee on their insistence that the choreic movements had to be "of at least moderate severity" to enable that symptom to be accounted. He said that occasionally one saw profound chorea, yet with the paralytic aspect so overriding the other that the movements were almost unnoticed. Distinction from habit spasm should cause no difficulty, and if there was any doubt the handwriting test helped. Sometimes profound psychological changes overshadowed the other symptoms. It was well known that only a small proportion of chorea sufferers developed other rheumatic manifestations. There was usually no fever, leucocytosis or raised erythrocyte sedimentation rate unless carditis developed.

Dr. Arden then said that rheumatic nodules hardly counted as a diagnostic feature. They were rare in Australia. Without knowing the exact figures he would say that only somewhere between 1% and 5% of child patients had them at any stage. They seldom appeared at the onset of the disease, and they almost always occurred in children who were in other ways severely affected. *Erythema marginatum* similarly was not common and meant little on its own. *Erythema nodosum* was not regarded as a specifically rheumatic manifestation, although the swellings on the legs were sometimes accompanied by fever and joint pains. Fever was not as a rule a prominent symptom, and unless there was pericarditis the temperature was seldom high. Its response to aspirin was often as striking as the disappearance of the joint symptoms. Perhaps the most important fever in rheumatism was the prolonged low-grade pyrexia that could accompany the subacute attack.

With regard to the raised erythrocyte sedimentation rate, Dr. Arden said that they must never forget that anæmia with a low red cell count, or more specifically a reduced hæmatocrit reading, would give fictitiously high erythrocyte sedimentation rate results. Normal readings helped to exclude rheumatism when one met anxious mothers whose children had "growing pains". Dr. Arden stressed that no importance attached to an elevated sedimentation rate if it was found when the patient was feverish. It was not by itself a diagnostic test for rheumatic fever, but only indicated an infective process. The erythrocyte sedimenta-

tion rate might improve and even return to normal in the presence of congestive heart failure. Evidence of preceding streptococcal infection should be sought as a routine procedure by taking a preliminary throat swab and examining it for β-hæmolytic streptococci. A high antistreptolysin O titre meant recent streptococcal infection, but did not indicate any specific variety of streptococcus. The titre reached its maximum level about four weeks after the infection, and then fell. Late tests were therefore useless. The procedure was not always reliable for young children, and might not achieve the recognized titre of 200 even after known streptococcal infections. In doubtful cases, antistreptolysin O readings of less than 50 virtually excluded rheumatic fever. That was perhaps the chief value of the test. Electrocardiographic changes might go beyond an increase in the *P-R* interval. Extension of the *Q-T* interval was equally significant. Also, an inverted *T* wave in any two of the three standard leads suggested myocardial involvement. However, one was often disappointed with electrocardiographic tracings, being presented with normal electrocardiograms by children with obvious mitral valve involvement. The importance attached to evidence of preceding attacks emphasized the frequency of subsequent episodes of rheumatic fever. They were particularly prone to occur in the first three years after the initial illness.

Dr. Arden then said that the foregoing list of five major and five minor manifestations of rheumatic fever was not quite the whole story. The family history was important, and should always be taken into account in the doubtful case. The tendency to develop rheumatic fever was a familial characteristic, and seemingly both heredity and proximity operated. Relatives living away from home were more liable to the disease than were strangers; but relatives in contact with a patient showed a higher incidence still. Growing pains were a frequent complaint and most difficult to assess. They were usually not rheumatic, being generally located in muscles rather than in joints. If they occurred only at night, the pains were probably due to muscle fatigue, circulatory changes or orthopædic defects; but in all cases they needed careful checking and examination. General symptoms such as pallor, fatigue, anorexia, weight loss and epistaxis were daily complaints in the consulting room. They were not, of course, specific symptoms of rheumatic fever, but had to be treated with suspicion. Cases which later presented carditis and no joint signs might begin in that way. Tachycardia, disproportionate to the temperature and in the absence of emotional upheaval, was an important sign. It was always a good plan to have the sleeping pulse rate recorded by the night nurse or parents. Dr. Arden thought that perhaps the laboratory staff might help a little further. They could estimate the amount of C-reactive protein in the serum. That protein formed a precipitate with the C polysaccharide of the pneumococcus, and appeared in the blood only in the presence of an inflammatory reaction. It was a very sensitive indicator of rheumatic activity, provided that other pathological processes could be excluded. However, it was no better than estimation of the erythrocyte sedimentation rate, and more difficult to perform. There was also a significant elevation of serum α globulin level and of fibrinogen level in the acute phase of rheumatic fever, with a depression of the serum albumin level. There was evidence that the serum α globulin level was slower to return to normal than either the erythrocyte sedimentation rate or the C-reactive protein level, and that it was sometimes raised when the other tests gave normal results. It had been held to be the most sensitive of the three tests, but it was still a non-specific one. Unfortunately the ordinary blood count was not helpful, revealing no more than slight to moderate anæmia, and leucocytosis in the 10,000 to 12,000 per cubic millimetre range.

Dr. Arden finally said that they were faced with a disease process which could express itself in a variety of ways. As a rule symptoms and signs were numerous enough and clear enough to make the diagnosis straightforward, but there would always be some difficult borderline cases. In those, the most careful sifting of all the evidence was essential. He begged that the child be kept in bed while the doctor was making up his mind.

Obesity and Anorexia.

Professor J. H. Sheldon (Wolverhampton, England) read two papers which he described as opposite aspects of the same problem—one on "Obesity", the other on "Anorexia".

Cardiac Invalidism.

Sir Alexander Murphy (Brisbane) read a paper entitled "The Prevention and Treatment of Cardiac Invalidism".

After Gastrectomy.

Dr. Evan Thomson (Brisbane) read a paper on the results of surgery in peptic ulcer. He said that partial gastrectomy for chronic peptic ulcer was one of the most satisfactory operations in surgery, provided that it was carefully performed on the correct patients. The immediate mortality rates had been stated by Ogilvie to be 0·2% to 0·5% for short-circuit operations, 2% to 3% in gastrectomy for ulcer, and 5% to 8% in secondary operations. Many experienced surgeons claimed lower mortality rates. The rate was higher in patients in the older groups, perhaps 10% to 15% in patients aged over fifty years. Dr. Thomson referred to Ogilvie's assessment of the end results of gastrectomy, and pointed out that in male patients particularly, earning capacity and fitness for heavy work were important. Most patients could return to their previous occupation or to equally heavy work, and few were unfit to earn their living. Turning to the complications of gastrectomy, Dr. Thomson said first that respiratory complications were not uncommon, and infective complications, particularly subphrenic abscess, caused concern at times. Necrotizing or pseudo-membranous enteritis was the cause of an occasional tragedy. The condition affected the small bowel predominantly, though the large bowel, the stomach and even the œsophagus might be involved. The condition arose suddenly during the first ten days after operation, with abdominal pain, vomiting, sweating and signs of severe circulatory failure. Fever was usually pronounced, and the patient might pass offensive, watery, green-yellow stools. Death from overwhelming dehydration and toxæmia might occur within four to forty-eight hours. Recovery was without stricture formation. The condition was at present regarded as due to resistant strains of *Staphylococcus aureus*, and it had been thought that the administration of antibiotics, particularly those with a broad spectrum, contributed by reducing the population of normal inhibitory intestinal organisms. That was not proven. Another complication was hæmorrhage from the anastomotic suture lines; with careful technique it was uncommon, and almost never demanded interference. Leakage from the gastro-jejunal suture line was also uncommon after operations for ulcer; it occurred more frequently in older patients and in those with carcinoma. A classical complication was the "blown duodenal stump", which might result from inadequate closure, from sloughing of the duodenal wall owing to inadequate blood supply, and from digestion of the suture line by pancreatic ferments. Dr. Thomson thought that the important factor must be obstruction of the afferent jejunal limb. Obstruction from œdema might also occur where the jejunum left the anastomosis; excessive aspirations would result. Gastrectomy, particularly the ante-colic type of operation, might be complicated by mechanical obstruction of both afferent and efferent limbs of jejunum. The ante-colic operation left a gap between the anastomosis and the transverse colon, and the retro-colic operation might leave a gap behind the afferent limb of jejunum. Herniations of gut or of omentum could occur through those spaces. Strangulation, gangrene and perforation, particularly of the afferent loop, might occur. Diagnosis was frequently difficult, and the mortality was considerable. Acute pancreatitis might complicate the post-operative course. It was uncommon, but important, because it carried a high mortality.

Dr. Thomson went on to discuss the remote sequelæ of gastrectomy, stressing the fact that they would affect only a minority of patients to any significant degree. He said that that was surprising, but true. Also the effects could vary from the most negligible to the most profound, even after similar operations by the same surgeon. One factor was the amount of stomach remaining; but the personality of the patient played a major part. To understand some of the sequelæ, it was necessary to note what was involved in gastrectomy. First, the gastric capacity was reduced, so that the total intake of food was cut down unless more frequent meals were taken. Over-eating might produce symptoms from distension of the gastric remnant. Secondly, the production of gastric enzymes was also reduced. Thirdly, the production of hydrochloric acid was intentionally reduced; but that interfered with absorption of iron. Fourthly, production of the intrinsic factor was interfered with; but that was unimportant unless total gastrectomy had been performed. In the fifth place, the pyloric sphincter was removed, and that disability was not remedied by the use of any of the suggested valvular mechanisms. In the sixth place, the rate of passage of food through the intestine was greatly accelerated, and diarrhœa might result. A consequence of the more rapid gastric emptying and intestinal "hurry" was that food, instead of being mixed in the duodenum with the bile and pancreatic fluids which its

very presence in the stomach produced, passed down the duodenum followed considerably later by those digestive juices which it never met. That was one of the reasons why frequent feeding was essential after gastrectomy. Each meal would then in the jejunum meet and mix with the bile and pancreatic fluids called forth by the previous meal. Moreover, after gastrectomy pancreatic juices were no longer secreted at the right time. Acid stimulation of the duodenum was absent, and after a Pólya procedure food did not enter the duodenum at all. The humoral mechanisms were grossly disturbed. In some cases of severe nutritional deficiency, considerable improvement followed conversion of a Pólya type operation to a Billroth I procedure. Another result of side-tracking of the duodenum and the proximal part of the jejunum was interference with iron absorption, most of which took place from that part of the intestine; the raised pH in the bowel was another factor. In the Pólya procedure an intestinal cul-de-sac was created from duodenum and a variable length of jejunum, and various untoward results might ensue.

Dr. Thomson then discussed the particular conditions that might occur after gastrectomy. Referring first to recurrent ulceration, he said that it was of uncommon occurrence. It was well to remember that hæmorrhage, perforation and fistula into the colon were all seen from time to time. Turning to nutritional disturbances, Dr. Thomson first mentioned primary Addisonian anæmia, which inevitably followed total gastrectomy, so that replacement therapy was always needed. He said that Addisonian anæmia was no more common after partial gastrectomy for ulcer than in the general population. Iron deficiency anæmia occurred in a significant number of cases, and women suffered more than men after gastrectomy. Iron was absorbed from an acid medium, and predominantly from the duodenum and the proximal part of the jejunum. Reduced acid level and side-tracking of the duodenum seriously upset that mechanism. Iron deficiency anæmia was commoner after operations for gastric than for duodenal ulcer, and less frequent after the Billroth I procedure, which maintained the duodenum in alimentary continuity. For that reason that procedure was performed whenever possible, and was the operation of choice for all patients with gastric ulcers, particularly in women. In women it was advisable to avoid putting the duodenum out of action, at least during the years of menstrual function. It was wise to instruct all patients after gastrectomy to report at least once a year for investigation of the hæmoglobin value, or alternatively to take iron for at least one month in each year. Failure to gain weight or loss of weight after operation constituted an important problem. After discussing the ætiological factors involved, Dr. Thomson referred to the ways in which the situation might be remedied. He said that first of all the patient was instructed to eat extra fat and protein, and protein preparations and reinforced milk were administered. Food should be taken more frequently, at least six times a day. Intestinal "hurry" might be reduced by the administration of hexamethonium bromide. Enzyme replacement therapy might be used. Finally, reoperation might be necessary. Another deficiency that might occur was that of vitamins, particularly the *B* complex. Patients should be advised to take extra vitamin *B* in the form of "Marmite" or some similar preparation, preferably continuously, but otherwise for at least one month in every year. Some patients complained of lack of energy, which might be unassociated with anæmia, loss of weight or vitamin deficiencies. Diarrhœa occasionally occurred; hexamethonium might be of help. Specific food idiosyncrasies might fellow gastrectomy; foods involved were milk, cream and eggs, and alcohol might not be tolerated. Connected with the nutritional disturbances was an increased risk of pulmonary tuberculosis, both from apparently fresh infections and from reactivation of quiescent lesions.

Discussing the post-gastrectomy, or "dumping", syndrome, Dr. Thomson first advocated the abandoning of the term "dumping", as providing an incomplete explanation for the several distinct groups of symptoms that might be experienced. He then described the various manifestations of the syndrome. The first was vomiting of bile, which might occur alone, or in association with other postprandial symptoms. It usually arose fairly early after operation, and tended to decrease with the passage of time; it occurred at unpredictable intervals and for no obvious reason. Some of the patients might also suffer from an œsophageal hiatus hernia of sliding type, with incompetence of the cardio-œsophageal valvular mechanism. In the remainder, and in the true "bilious vomiter", the cause probably lay in the afferent limb; indeed, the condition had been regarded as one variety of "afferent loop syndrome". The second condition described by Dr. Thomson was the late postprandial syndrome, which he said was very uncommon. It was generally agreed that the symptoms were due to hypoglycæmia; they were certainly those of the hypoglycæmic state. Dr. Thomson described the mechanism of their production, and said that the syndrome arose at a variable period after operation, possibly depending on the frequency of feeding; it rarely lasted for more than five years after operation. The early postprandial syndrome was much more common; it presented the greatest post-gastrectomy problem. It usually arose soon after gastrectomy, but its onset might be delayed even for years. In most cases the tendency was towards improvement, but in the more severe cases the symptoms might continue indefinitely. Symptoms were usually aggravated by over-eating, and many patients learned how much they could safely eat. Excess sugar might also precipitate symptoms, which commonly consisted in a sensation of epigastric fullness, nausea, palpitations, sweating and a feeling of warmth. About half the patients also suffered from bilious vomiting, and failure to gain weight was common. Dr. Thomson discussed the ætiological background, and then quoted the following advice which he gave to all patients after gastrectomy:

1. You are no longer on a diet. You may eat anything.

2. You must always eat frequently, at least six times a day. (This means merely that morning tea, afternoon tea and supper should be a regular habit.)

3. Eat just up to your capacity. Discomfort may follow overloading your stomach.

4. Eat your main meals dry without drinking at the same time.

5. Avoid taking too much sugar and avoid food such as milk etc. if you find you react unfavourably.

6. If you are uncomfortable after meals, lie down.

7. Eat rather more fat and protein foods than the average. If necessary take some protein preparation as well.

8. Eat extra "Marmite" and report each year for a blood count or take some iron at least one month in each year.

DR. T. M. GREENAWAY (Sydney), in opening the discussion, said that he was in complete agreement with all that had been said. He thought that he had seen better results from removing more stomach rather than less, but the surgery must be good, and the follow-up good. Although primary anæmia was rare after gastrectomy, he thought that vitamin B_{12} therapy still had some place in atrophic gastritis by its action on the mucosa.

DR. H. HARRIS (Broken Hill) mentioned three patients whom he had examined with peptic ulceration, duodenal in two and gastric in one, and who developed pulmonary tuberculosis. He wondered whether the tuberculosis was actually present first and caused the ulcers, and whether in fact the ulcers were tuberculous, and the pulmonary lesions had only appeared subsequently.

Dr. Greenaway thought that the tuberculous state was not particularly related to operation, and reminded the listeners that tuberculosis was also common in the later years of life.

DR. J. BELL (Lismore) said that he had observed that peptic ulceration seemed to be largely a male prerogative, and also that it was rare in farmers. He thought that the latter observation might be due to the fact that the average farmer spontaneously adopted the dietary regime of a healthy schoolboy.

DR. P. LESLIE (Toowoomba) asked about the intramuscular use of iron in relation to iron-deficiency anæmias.

Dr. Greenaway agreed that it was good, but said that it was not so painless as was thought.

Emergencies in General Practice.

The New South Wales Faculty of the College of General Practitioners contributed a symposium on "Emergencies in General Practice". The speakers were Dr. R. J. Jackson (Armidale), Dr. A. P. Millar (Bexley) and Dr. A. Rumore (Punchbowl).

DR. R. J. JACKSON said that at the outset it was desirable to define exactly what one understood by "emergencies". They were taken to be those unexpected occurrences in one's work which were better defined, perhaps, as unforeseen sets of circumstances which called for immediate action. It had to be stated that the accent in that definition was on the unexpectedness of the occurrence. The measure of a general practitioner's all-round proficiency in his profes-

sional work was the infrequency of the unexpected occurrence. "More mistakes are made by not looking than by not knowing" was one of the most corrective of the aphorisms of medical training and practice. The acquisition of the all-seeing eye and the correct deduction from what one saw with that "X-ray eye" were amongst the ceaseless aims of general practice. Emergencies therefore were subjective, not universal. Occurrences that presented unexpectedly in the treatment of a disease or in the management of a patient might be met quite calmly by one doctor; whereas, when they similarly confronted another, they might cause hesitancy of decision and uncertainty of action, resulting in disaster.

Dr. Jackson went on to say that "the moderator of the panel" had allotted to him a sample emergency, with appropriate reference to country conditions, in each of two of the many fields of general practice. The first, an obstetrical case, told simply and slowly the old, old story which busy doctors forgot so soon—the story of the necessity in practice for painstaking methodical care to diagnose correctly—more mistakes were made by not looking than by not knowing. It was with mixed feelings of diffidence, relief at the outcome and keen interest in the views that might be forthcoming from members of the audience on the subjects of version and breech presentation, that he presented the following case of Mrs. M. She was aged twenty-eight years and had been married happily for four years, when she was diagnosed as pregnant on May 31, 1955; her expected date of confinement was calculated to be November 23; the first day of her last mentrual period was stated to have been February 13. Other than much inverted nipples, which had been treated along lines already described by Dr. Stuart Patterson, no abnormality was found on examination. Her blood group and Rh factor were determined. Regular ante-natal check examinations were carried out, and a smooth period of pregnancy was observed.

At midnight on December 5 she was admitted to hospital, where the ward examination report was as follows: "Position vertex, fœtal heart sounds normal, head engaged, no pains and no dilatation of the cervix." Over the following seven days uterine contractions began and continued spasmodically; a medical induction of labour in accordance with the teaching of Professor B. T. Mayes in his "Textbook of Obstetrics" was carried out, with no immediate result. On the morning of December 13 the assistance of a colleague on the hospital staff was sought. The result of that consultation (at which the cervix was found to be almost fully dilated with the presenting part well down, and the membranes ruptured during examination) was the decision to allow the birth to follow a natural course. During the afternoon, after a check examination of the baby's position and heart sounds, an X-ray film was taken. The report was: "Breech presentation with extended legs." That was the unexpected occurrence for him—a *primipara*, not so young as one could have wished, with taut abdominal muscles, post-maturity, and fatigued by the physical effort of a long-drawn-out labour and the mental strain of hope deferred day after day, with the baby's position as shown in the film. Dr. Jackson said that a further consultation with his colleague was followed by the decision to allow the delivery to proceed naturally—not, of course, without apprehension on their part. The possible difficulty they could have with the after-coming head was vividly in their thoughts. Professor Mayes had clearly expressed that state of mind: "The inability to assess early enough in labour a disproportion between head and pelvis is due to the very nature of breech presentation where the head and pelvis are initially 'poles apart'. It is the one oustanding doubt persistently present in the accoucheur's mind which makes his approach to a breech delivery always tinged with an element of anxiety no matter what his experiences may have been." That evening, under general anæsthesia, Mrs. M. was successfully delivered of a baby weighing nine pounds five ounces. The immediate emergency had come to an end. Subsequent worries, however, were moderately severe maternal pyrexia for four days, hæmorrhage of the newborn in the infant and, later, wry neck, which responded to stretching exercises.

Dr. Jackson went on to say that the second case allotted to him as an emergency in general practice was a good instance of successful cooperation in the fields of general and special practice; it illustrated early diagnosis, effective treatment and specialist availability. To him, the general practitioner, the exact diagnosis of that patient's condition provided the emergency—the unexpected occurrence. On January 11, 1952, a boy, aged seven years, living in a country district of New England, was brought in by his parents because they had recently observed peculiarity in his walking. His right leg appeared to swing outward so that the right knee knocked against the left; also the right foot "flopped" down with each step taken instead of being purposefully placed down flat as in normal gait. He had had no previous relevant illness, and the family—parents and two younger children—were all healthy. Abnormalities revealed on examination were: (i) a slight adduction of the right thigh so that the right femoral trochanter was a little more prominent than the left; (ii) Babinski's sign on the right side; (iii) disturbance of sensation of the right lower limb; (iv) weakness of the *extensor digitorum longus*, the *tibialis anterior*, the peronei and the glutei on the right side. An X-ray film of the right hip joint was reported as showing no abnormality. A progress examination five days later accented the appreciable development of his leading symptoms. The clinical note made then was: "The boy walked into the room with a more marked throwing out of the right leg which traversed a greater arc and then flopped more heavily onto the floor. When undressing himself he used his right hand very little, fumbling his braces and buttons with his left hand. He was more irritable and tended to weep." (Previously he had been eager to help, interested and cooperative.) The signs were much the same; his sensation responses were not consistent. In his fundi, with the pupils not diagnostically dilated, no abnormality was seen.

Dr. Jackson said that the incomplete conclusion at that point was that he had a central nervous system lesion which was progressing at an appreciably increasing rate. He was, in fact, a "diagnostic emergency". The unexpectedness of the final diagnosis was to be found in the report of the neurosurgeon to whom he was referred. Its more relevant sections were the following:

> Definite signs of some large space-occupying lesion in his left cerebral hemisphere, double papilloedema, spastic ataxic hemiplegia of the right limbs. Blood count was normal, as was the Casoni test, but the complement test for hydatids was strongly positive. Exploratory craniotomy was followed by the removal of a very large hydatid cyst with about twenty daughter cysts outside it from the left parietal region near the middle line. The progress should be good.

During the past four and a half years the boy had remained under family doctor and specialist watchful observation and had continued to develop normally.

DR. A. P. MILLAR discussed the treatment of cardiac asthma. He said that the immediate treatment was the administration of morphine in suitable doses, as that relieved the immediate symptoms satisfactorily. The addition of hyoscine produced a satisfying and restful sleep. For those who vomited with morphine, the combination of pethidine with hyoscine had proved satisfactory. The next step in treatment depended on the ætiology. Those patients who were hypertensive were benefited by hexamethonium given intramuscularly in dosage sufficient to lower the systolic blood pressure to about 150 to 170 millimetres of mercury. Lower levels were usually associated with giddiness. For cases of cadiac asthma associated with irregular rhythms, digitalis given intramuscularly had no equal. It slowed the heart rate, improved the circulatory imbalance and gave an early start to correct treatment. Cases of cardiac asthma due to coronary occlusion were managed as for the causative lesion. Patients whose dyspnœa was precipitated by paroxysmal tachycardia could be managed with good results if an intelligent course of treatment was followed. The first step was to apply carotid sinus pressure. Morphine sometimes stopped the attacks. Other drugs of value included "Mecholyl", digitalis, quinidine and "Promesty1". They might be used intravenously if reasonable care and patience were exercised. For those patients whose lesion was coronary sclerosis, aminophylline given intravenously was of help as it improved the coronary blood supply.

DR. A. RUMORE discussed cardiac arrest (acute circulatory arrest). He said that of all the emergencies with which the general practitioner was likely to be faced, the most terrifying one was surely cardiac arrest during anæsthesia. It occurred very rarely, the incidence being one in 500 to one in 5000 operations. It might occur in children or in young, healthy adults undergoing operations of small magnitude. The recovery rate was 30%, and recovery always depended on the management of the patient by the practitioners present and on the equipment available. Irreversible cerebral changes would occur after two or three minutes of anoxia, so that there was little time to waste and immediate action was necessary.

Dr. Rumore went on to say that anoxia was the most important if not the only cause of cardiac arrest. It seemed

that anoxia sensitized a vasovagal reflex causing cardiac arrest. Reflex vagal stimulation might be precipitated by an intravenous pyelographic investigation, by bronchography, by intratracheal intubation, by jugular punctures, by "Pentothal" induction of anæsthesia, and by surgical incisions or manipulations made before the patient was properly anæsthetized. Chloroform, ethyl chloride and cyclopropane had a direct action on the myocardium and might also cause cardiac arrest. Most of these cases occurred during the induction of anæsthesia or when the patient was recovering from the anæsthetic, and therefore the surgeon would not be in a position to make a diagnosis. The immediate recognition of the condition by the anæsthetist was absolutely essential. Acute circulatory arrest existed when the carotid pulse was absent. Immediately the heart sounds were auscultated, and if they were absent, two essential steps were undertaken without hesitation.

The first step, undertaken by the surgeon, was intrathoracic cardiac massage, which was five times more effective than intraabdominal massage. The left pleural cavity was opened by a transverse incision just lateral to the sternum in and down through the fourth or fifth intercostal space. The adjacent costal cartilages might be cut if necessary. The heart was grasped in the right hand with the thumb held anteriorly, and rhythmic, firm, but not violent compressions were carried out at a rate of 50 to 60 per minute. The second step, which was also undertaken immediately the diagnosis had been made, was performed by the anæsthetist. It consisted of adequately ventilating the lungs with oxygen. An intratracheal tube was passed into the larynx with a laryngoscope and attached to an anæsthetic machine. The bag was filled with oxygen, and intermittent compression was performed. If neither a laryngoscope nor an anæsthetic machine was available, a simple, inexpensive piece of apparatus was satisfactory; it consisted of a squeeze bag with an outlet to fit an oxygen cylinder and tube connexion to a rubber face mask. That was all that need be done for the first two or three minutes. Dr. Rumore pointed out that no drug of any kind had been injected into the circulation until effective cardiac massage and adequate lung ventilation had been affected.

Dr. Rumore went on to say that in 88% of cases there was ventricular standstill. When that occurred, after two to three minutes a one in 1000 solution of adrenaline, one cubic centimetre diluted with nine cubic centimetres of normal saline, was injected into the left auricle, and massage was continued for another two to three minutes. The injection could be repeated at intervals to a total dosage of five cubic centimetres of one in 1000 adrenaline solution diluted with saline. Life might be maintained in that way for up to one hour, with recovery. In 12% of cases ventricular fibrillation occurred. The use of adrenaline was contraindicated in that condition, and a myocardial depressant was injected into the left auricle. That was a 1% solution of procaine, which was given in 10 cubic centimetre amounts. The administration was repeated at intervals to a total of 50 cubic centimetres of 1% procaine solution as a single dose in an adult. If a defibrillator was available, it might be applied directly to the heart and repeated electric shocks administered. An electric pacemaker, which consisted of a portable machine that could be applied externally to the chest wall before thoracotomy, was valuable in cases of ventricular standstill. It was not effective in cases of ventricular fibrillation. It was also useful in restarting the heart in cases in which cardiac arrest recurred after cardiac massage.

Dr. Rumore then said that, the heart having been restarted, the chest was closed by through-and-through sutures with or without underwater drainage. The patient was still in grave danger of death from cerebral œdema resulting from cerebral hypoxia. The hypoxia caused dilatation of the cerebral capillaries. That might occur when the hypoxia was due to causes other than cardiac arrest, such as *asphyxia neonatorum*, coma, shock, hypotension due to drugs *et cetera*. The capillary damage caused protein to pass out into the extracellular fluid, which produced a rise in osmotic pressure; that brought about more cerebral œdema, which in turn interfered with oxygenation of the damaged brain cells and caused permanent cerebral damage. Thus dehydration might break that vicious cycle. Clinically, there would be a history of an anoxic or hypoxic episode, and also coma, stertorous breathing, hyperpyrexia, restlessness and fits, and the patient might die, or might recover with hemiparesis, incontinence of urine and fæces or defects of vision. Dehydrating agents, by increasing the crystalloid osmotic pressure of the plasma, drew water into the circulation from the interstitial fluid, and the intracellular fluid passed into the extracellular spaces. Glucose solution (50%) was not suitable, because it passed through the arachnoid membrane and caused a rise in the osmotic pressure of the cerebro-spinal fluid. Sodium chloride solution (30%) was too toxic. Concentrated human serum might cause serum jaundice. Sucrose solution (50%) was not stored in the tissues and produced considerable diuresis. The procedure was to inject 40 cubic centimetres of 50% sucrose solution intravenously and wait for fifteen minutes. Some improvement would be seen, and the same dose might be repeated. Half the dose or the same dose might be repeated in half an hour. A transfusion of 10% dextran solution might then be started. The patient was nursed in a 7° downward position, oxygen was administered continuously, antibiotics were given, fluids were restricted, and the blood pressure was maintained by nor-adrenaline if necessary. Tracheotomy might be required for secretions in the larynx, and gastric suction, stomach feeding, the administration of vitamins and blood transfusion might be necessary; the patient might need to be in a respirator. Over-sedation had to be avoided. The administration of carbon dioxide for short periods might be helpful. Stellate ganglion block might improve the cerebral circulation, and hypothermia might help patients with hyperpyrexia.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

RESPONSIBILITY OF HOSPITAL BOARDS.[1]

[From the *Australasian Medical Gazette*, March 15, 1886.]

AT the last monthly meeting of the Directors of Sydney Hospital a letter was received from a former house surgeon Dr. Newmarch now of Bowral in which he asked the authorities of the Hospital to repay him £32.9.8 the amount of his Attorneys Costs incurred in his defence of a late action brought against him by a patient of that Institution. The action as our readers will remember resulted in a verdict in Dr. Newmarchs favour without his being called on for his defence, the plaintiffs Attorney apologizing for having brought what proved to be such an unjustifiable action. The Directors with one or two miserable exceptions received the letter in the fair and liberal spirit which might have been expected of them one generously suggesting that, if no funds belonging to the Institution were available for the purpose the Directors should put their hands in their own pockets to refund the amount.

There can be no doubt that if not legally responsible the governing body is morally bound to repay the expense to which one of their paid officers (who has properly done his duty) may have been put in defending an action brought against him as a consequence of Circumstances which have originated in the performance of his ordinary duty.

Correspondence.

MEDICAL BENEFITS.

SIR: Your anonymous correspondent of February 16 complains of delay in receiving from a patient a Medical Benefits Fund cheque, and brands as unsatisfactory to doctors and profitable to the fund the system of posting to the patients cheques made payable to doctors. This fund service to doctors and their patients is neither satisfactory nor profitable to the fund.

A very potent safeguard against abuse of the Australian national health scheme is the rule that medical benefits paid must not exceed 90% of the doctor's fee. The settlement of claims for benefit is between the contracting subscriber and his medical benefits fund on the basis of his doctor's itemized account. If the patient has been unable to pay the doctor beforehand, the fund sends to the patient for use by him when paying the doctor a benefit cheque drawn in the

[1] From the original in the Mitchell Library, Sydney.

doctor's favour. It is expected that the patient with the assistance of the fund cheque will then pay the doctor in full, and that the doctor accepts the responsibility of collecting his fees.

The Commonwealth Department of Health wisely put a stop to the practice adopted by some medical funds of settling claims in bulk direct with doctors, thus safeguarding not only the financial stability of the funds concerned and the Commonwealth health scheme, but also the integrity of the medical profession.

The Medical Benefits Fund looks to the medical profession and to the general public for criticisms and for suggestions, and this opportunity is taken to invite interested subscribers to THE MEDICAL JOURNAL OF AUSTRALIA to communicate direct with the fund on matters which may give them concern from time to time. Published anonymous letters as a matter of policy are not usually answered.

Yours, etc.,

J. F. CADE,
General Manager, Medical Benefits Fund of Australia.

32 Jamison Street,
Sydney,
February 21, 1957.

FACTORS AFFECTING THE MORTALITY OF SMALL-BOWEL OBSTRUCTION.

SIR: I should be grateful if you would allow me to make a few comments concerning Mr. Neville Davis's letter of January 16. In general I agree with Mr. Davis's quotation from Wangensteen's book, and nothing in it is contrary to what I have written.

I do not agree with Mr. Rodney Smith or with any other surgeon who advocates a time limit as a decisive factor in deciding on operation. Every case has to be considered according to its clinical condition and one's estimate of the probable pathological changes, quite irrespective of any arbitrary period of time. It should be apparent from what I have already written that I agree with the first of Mr. Smith's two criteria necessary to justify delay in operating, although I prefer to put it a little differentaly—namely: "That the only safe attitude is to operate in any case in which a non-resolving strangulation cannot be excluded." As to his second criterion, there is no doubt whatever in my mind, and, I should think, in the mind of anyone experienced in intestinal intubation, that the risk of delay resulting in gangrene of bowel is much less than the risk of intervening while the patient is grossly affected by dehydration, electrolyte imbalance and intestinal dilatation. The more intestine I inspect at operation, following a period of suction used in the manner I advocate, the more I feel that the dangers of free perforation or change from reversible to irreversible vascular changes during the establishment of some decompression are not nearly as great as I had earlier thought to be the case.

The sixteen-year-old girl whom I treated, and referred to by Mr. Davis, is the same case that was quoted in my paper of November 3, 1956, in this journal. On rereading it I find that, not thinking it would be of any particular importance to the reader, I did not make it clear that this girl was operated on by another as a case of acute appendicitis and through a McBurney incision, and did not come into my care until fourteen days after operation—that is, on the day that Miller-Abbott intubation was commenced. I make no apologies for the prolonged intubation. It, with resuscitation, made a tremendous difference to her general condition, and when it appeared that a non-resolving closed loop was present operation was performed. I again find that I did not make it clear that, when I performed this operation, I established that the original diagnosis had been incorrect, and that she was in reality suffering from an extensive pelvic peritonitis with abscess formation resulting from salpingitis. Enterostomy with drainage of the abscess was the correct treatment. The prolonged fæcal fistula caused me no concern, as the development of such in similar cases often proves of great benefit to the patient, though perhaps disconcerting to some surgeons. In this case many surgeons may have operated earlier, but with this treatment she was alive and well seven years later.

I feel Mr. Davis's remark that "when operation was finally performed only an enterostomy was possible" is unfortunate. It is not right to imply that enterostomy is an inferior method of treatment. It is not often that enterostomy is called for, but when it is, it is not being used as a last resort. It is being used, as it was in this case, as the proper treatment for that particular patient. The prime object in operating on a sick patient is to save his life, not necessarily to restore him anatomically to normal.

I feel that enterostomy as has been used in the past often failed, not because there was anything essentially wrong with enterostomy as such, but because it was not appreciated that such did not decompress the bowel above the most proximal block, and also probably in the days when enterostomy was a popular method of treatment, resuscitation methods were not nearly so satisfactory.

Mr. Davis further states: "In Mr. Gale's skilled hands there is no mortality with post-operative obstruction." I made two statements. Firstly: "This day I asked the surgical registrars of the Geelong Hospital how many cases during the last year of early post-operative obstruction under all surgeons had required operation after intestinal intubation, continuous suction, and intravenous therapy were used in the manner I advocate. The answer was none." The second statement was: "This day I asked the pathologist of the Geelong Hospital, Dr. V. Plueckhahn, who three years ago instituted post-mortem examination on all deaths, how many cases had occurred in which early post-operative obstruction was treated by the same methods, and in the manner I advocate either alone or with operation. The answer was none."

The first statement is in the process of being checked, and the second statement has been confirmed by check of post-mortem records of all deaths under all surgeons at the Geelong Hospital during the last three years. The statement not only refers to my personal cases, but to those of all other surgeons on the hospital staff.

These results apply to cases treated in the manner I have repeatedly advocated, and the failure of any surgeon to reproduce them, whether he be Wangensteen, Rodney Smith or Neville Davis, is due to failure to use the methods as they should be used. A professional statistician may not say the figures of the Geelong Hospital are technically statistically significant, but clinically they are to me. It is in the hands of any surgeon with access to clinical material to prove or disprove the correctness of what I have stated.

Yours, etc.,

CHARLES GALE.

T. and G. Building,
Geelong,
Victoria.
February 20, 1957.

[This correspondence is now closed.—EDITOR.]

CONGENITAL MALFORMATIONS AND MATERNAL RUBELLA.

SIR: Dr. Pitt's article is of considerable interest. He refers to a survey being organized at the University of Melbourne for the follow up of pregnant women with rubella. This is the most satisfactory method of assessing the risk of malformations from maternal rubella, and is the only way in which one can determine whether termination of pregnancy in these cases is justified.

Dr. Pitt commented on an earlier study made by myself on behalf of the Australian Red Cross Society, in which the available Australian figures were analysed with a view to determining which were the most dangerous months for the mother to contract rubella. The idea of the survey was to conserve the Red Cross Society's slender stocks of convalescent serum by giving it only to persons who had a real risk of having an affected child. The study was made at the beginning of a new epidemic, and there was no time available for any inquiry of the "prospective" type such as is being planned now at Melbourne. The "risk" figures obtained for the various months were needed as a guide as to the advisability of giving prophylaxis, and were very satisfactory for that purpose. The question of termination of pregnancy, with its medical indications complicated as they are by so many non-medical influences, was rightly considered to be no concern of the Red Cross Society and was not referred to in the article.

Many of Dr. Pitt's criticisms of the early statistics are justified, while some are unsound.

The retrospective method of inquiry must inevitably be inaccurate, owing to the omission from the statistics of numbers of patients who gave birth to normal children; and any survey where the conclusions may lead to termination of pregnancy in a patient should be of the "prospective" type.

The diagnosis of some of the cases of congenital heart disease is open to question, and a few should now be certainly regarded as normal. This, however, does not alter the fact that most of the congenital defects were clearly and accurately defined as far as space permitted. With regard to the rubella itself, it is probable that the diagnosis was far more accurate in the early studies than it is today. The early epidemics were severe, with high infectivity, and with an obvious clinical course. The present outbreak of rubella is mild, and many non-specific febrile rashes may easily be confused with it. Under present conditions, Dr. Pitt should not be satisfied with a diagnosis made by a resident medical officer to include cases in his series.

Lastly, there have been alterations in the character of the disease. The epidemics described by Gregg and Swan were followed by a high proportion of abnormalities. The present outbreak does not seem to have been particularly impressive in that regard. Future epidemics may not be so mild. It would seem advisable for the Melbourne workers to study a number of epidemics for their figures to have any meaning, and a long-term study of a decade or more may be necessary to demonstrate the true capacity of rubella outbreaks to produce congenital defects.

Yours, etc.,

IAN S. COLLINS.

The Medical Centre,
100 Carillon Avenue,
Newtown,
New South Wales.
February 23, 1957.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

PROGRAMME FOR APRIL, 1957.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that the following courses will be conducted in Sydney during April, 1957.

Course in the Basic Medical Sciences.

The following is the programme of a short course in the basic medical sciences embracing the subjects of anatomy, biochemistry, pathology, pharmacology and physiology, to be conducted over the period April 9 to July 16, 1957. It is not intended to be a comprehensive review, but rather to present certain outstanding features of these subjects. The selection of lecturers is largely based upon their particular interests. Its purpose is to provide a revision for established practitioners and to form an introductory series for practitioners who may later present as candidates for the various specialist diplomas. All lectures will begin at 8 p.m., and unless otherwise indicated will be held in the Post-Graduate Theatrette, 131 Macquarie Street, Sydney.

Programme.

The programme is as follows:

Tuesday, April 9: "Calcium Metabolism: Some Aspects of the Physiology of Parathyroids, Bones, Kidneys and Their Interrelationships", Dr. Lyal Watson. Thursday, April 11: "Fasciæ of the Neck and Their Clinical Significance", Dr. E. A. Hedberg. Tuesday, April 16: "Adrenal Cortex", Dr. Lyal Watson. Wednesday, April 17: "The Salivary Glands", Dr. J. M. Yeates. Tuesday, April 23: "Nervous and Chemical Control of Respiration", Dr. R. A. B. Holland. Wednesday, April 24: "The Nerve Supply of the Upper Limb", Dr. W. J. Burke. Tuesday, April 30: "The Uptake and Transfer of Gases in the Blood", Dr. R. A. B. Holland. Thursday, May 2: "The Venous Drainage of the Lower Limb", Dr. Justin Fleming. Tuesday, May 7: "Physiology of Valvular Heart Disease", Dr. P. I. Korner. Thursday, May 9: "The Mediastinum and Diaphragm", Dr. R. P. Melville. Tuesday, May 14: "Physiology of Congestive Heart Failure", Dr. P. I. Korner. Wednesday, May 15: "The Female Pelvis", Dr. R. D. Condon. Thursday, May 16: "Tranquillizing Drugs", Professor J. M. Robson, Visiting Professor of Pharmacology, Guy's Hospital Medical School, London (in the New Barn Theatre, New Medical School, University of Sydney). Tuesday, May 21: "Physiology of Gastro-Intestinal Motility", Dr. B. P. Billington. Thursday, May 23: "The Greater and Lesser Sac of the Peritoneum", Dr. P. J. Kenny. Tuesday, May 28: "Physiology of Gastro-Intestinal Secretion", Dr. B. P. Billington. Thursday, May 30: "Lymphatic Drainage of Breast, Stomach and Large Bowel", Dr. R. D. Condon. Tuesday, June 4: "The Reticular Formation", Dr. J. L. Allsop. Thursday, June 6: "The Anatomy of Inguinal and Femoral Hernia", Dr. R. P. Melville. Tuesday, June 11: "Autonomic Nervous System", Dr. J. L. Allsop. Thursday, June 13: "Energy Exchange in the Human Body", Dr. W. J. Hensley. Tuesday, June 18: "Acid Base Balance and its Regulation", Dr. W. J. Hensley. Thursday, June 20: "Nitrogen Metabolism in the Human Body", Professor J. L. Still. Tuesday, June 25: "Metabolic Antagonism and its Relation to Chemotherapy", Professor J. L. Still. Thursday, June 27: "Neuromuscular Blocking Drugs", Professor R. H. Thorp. Tuesday, July 2: "Newer Antibiotics", Professor R. H. Thorp. Thursday, July 4: "Newer Sedative Drugs", Professor R. H. Thorp. Tuesday, July 9: "New Information About Inflammation", Dr. V. J. McGovern. Thursday, July 11: "Problems of Renal Disease", Dr. Ernest Finckh. Tuesday, July 16: "Ossification", Mr. David A. Cameron.

The fee for attendance is £15 15s.

Tutorial Course Suitable for Primary F.R.A.C.S. Examination.

A tutorial course in anatomy, physiology and pathology will be held on Wednesday afternoons from April 10 to August 21, 1957. The fee for attendance is £26 5s.

Week-End Course in Radiological Diagnosis and Techniques.

A week-end course in radiological diagnosis and techniques will be held at the Royal Prince Alfred Hospital, Camperdown, on Saturday and Sunday, April 13 and 14, 1957, under the supervision of Dr. Alan R. Colwell. The programme is as follows:

Saturday, April 13: 9.30 a.m., "Fractures and Bone Injuries", Dr. McClements Callow; 11 a.m., "Œsophagus, Stomach and Duodenum", Dr. Alan R. Colwell; 2 p.m., "Small and Large Intestine", Dr. T. P. Loneragan; 3.30 p.m., "The Urinary Tract", Dr. Denis Wightman.

Sunday, April 14: 10 a.m., "The Biliary Tract", Dr. Brian E. Frecker; 11.30 a.m., "The Respiratory Tract", Dr. B. P. Anderson Stuart.

The fee for attendance is £3 3s.

Method of Enrolment.

Early written enrolment for these courses, enclosing remittance, should be made to the Course Secretary, The Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 4497-8. Telegraphic address: "Post-grad Sydney."

THE MELBOURNE MEDICAL POST-GRADUATE COMMITTEE.

PROGRAMME FOR APRIL, 1957.

Courses for Higher Qualifications.

THE Melbourne Medical Post-Graduate Committee will conduct a course in microbiology suitable for candidates for the M.D. and M.S. degrees and post-graduate diplomas. This will commence at the Bacteriology Department, University of Melbourne, on April 16, and continue on Tuesdays at 2.15 p.m. for about fifteen weeks. Enrolments, together with the fee of £9 9s., should be lodged with the Post-Graduate Committee by April 2.

Courses in anatomy, physiology, pathology and physics, which have already commenced at the University of Melbourne, will continue during April.

The Victorian Branch of the Ophthalmological Society of Australia (British Medical Association) will conduct a course in ophthalmic medicine and surgery and ocular pathology suitable for candidates for the D.O. Part II. The course will commence on April 8, and will consist of 70 to 80 lectures. These will be held in the late afternoon, chiefly on Tuesdays, Thursdays and Fridays, continuing till September. The fee for this course is £31 10s., and enrolments should be made through the Post-Graduate Committee on its special form.

The Victorian Mental Hygiene Authority's course in psychiatry has been arranged to commence on March 19 and to continue on Monday evenings till June 18, from

8 to 10 p.m. This year's course includes a symposium on some organic aspects of psychiatry. There is no fee, and those desiring to attend should communicate with the Chief Clinical Officer, Mental Health Research Institute, Poplar Street, Royal Park, N.2. Lectures will be held in the Training Centre, Royal Park Receiving House, Oak Street.

In May, courses in otolaryngology and radiodiagnosis will commence, and details will be announced shortly.

Country Courses.

Traralgon.—On April 6 and 7, at the Traralgon and District Hospital, the following lectures will be given: Saturday, April 6: 8 p.m., "Chronic Back Pain", Mr. J. Clarke McNeur; 8 p.m., "Electron Microscopy", Dr. John Hurley; "Diagnosis and Treatment of Psychosomatic Disorders", Dr. Bruce Robinson. Sunday, April 7: 10.30 a.m., "Infertility", Dr. G. Ley. The local secretary for this course is Dr. J. E. Joseph, 237 Princes Highway, Morwell. Fees are at the rate of 15s. per session, but those who have paid an annual subscription to the Post-Graduate Committee may attend without further fee.

Flinders Naval Depot.—On Wednesday, April 3, at 2.30 p.m., Sir Albert Coates will discuss "Surgical Problems in Survival". This is by arrangement with the Royal Australian Navy.

Overseas Lecturers.

Dr. Merrill C. Sosman, Professor of Radiology of Boston, United States of America, will visit Melbourne from April 7 to 11 and give the following lectures in the British Medical Association Hall at 8.15 p.m.: Monday, April 8, "The Accuracy and Reliability of the Röntgen Examination"; Thursday, April 11, "Röntgen Aspects of Heart Disease". Fees for the above lectures are 15s. each, but those who have paid an annual subscription to the Post-Graduate Committee may attend without further charge.

Dr. John Bromley and Dr. Whateley Davidson, of the Faculty of Radiologists, may visit Melbourne at the end of April for a few days. Details of any lectures they may give will be announced by the Post-Graduate Committee as soon as possible.

Sir James Paterson Ross, K.C.V.O., F.R.C.S., will visit Melbourne from April 23 to May 4 as the Sims Commonwealth Travelling Professor. Details of his programme may be obtained from the Royal Australasian College of Surgeons.

Dr. Geoffrey Organe, M.D., F.F.A.R.C.S., will visit Melbourne from April 19 to 28, under the sponsorship of the Australian Society of Anæsthetists. He will lecture in the Royal Australasian College of Surgeons lecture theatre at 8.15 p.m. on Wednesday, April 24, on "The Teaching and Practice of Anæsthesia". On Friday, April 26, at 1 p.m., he will attend the clinico-pathological meeting at the Royal Melbourne Hospital and speak on "Anæsthesia and the Anæsthetist".

Inquiries.

The address of the Melbourne Medical Post-Graduate Committee is 394 Albert Street, East Melbourne, C.2. Telephone: FB 2547-2548.

Naval, Military and Air Force.

APPOINTMENTS.

The following appointments, changes *et cetera* are promulgated in the *Commonwealth of Australia Gazette*, No. 5, of January 24, 1957.

Australian Military Forces.

Australian Regular Army.

Royal Australian Army Medical Corps (Medical).

3/40055 Captain G. A. Scott is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Southern Command), 9th November, 1956.

To be Temporary Lieutenant-Colonel, 23rd November, 1956: 3/40123 Major A. P. Hanway.

Citizen Military Forces.

Southern Command.

Royal Australian Army Medical Corps (Medical).—To be Major, 15th November, 1956: 6/15358 Captain C. S. Reid.

Central Command.

Royal Australian Army Medical Corps (Medical).—F4/1171 Honorary Captain R. L. Dow is appointed from the Reserve of Officers, and to be Captain (provisionally), 25th October, 1956. The provisional rank of 4/32054 Captain L. C. Hoff is confirmed.

Western Command.

Royal Australian Army Medical Corps (Medical).—The provisional appointments of the following officers are terminated, 29th September, 1956: Captains 5/26530 S. Shub, 5/26531 M. N. I. Walters and 5/45806 J. M. Lubich. To be Captains (provisionally), 30th September, 1956: 5/26530 Samuel Shub, 5/26531 Max Norman Isadore Walters and 5/45806 Joe Maurice Lubich.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps (Medical).

Northern Command.—To be Honorary Captain, 16th November, 1956: Margaret Wendy Hoskin.

Southern Command.—To be Honorary Captain, 8th October, 1956: Robert William Hill.

Northern Command.—To be Honorary Captain, 8th November, 1956: Rodney Carl Rogers.

Southern Command.—Honorary Captain K. L. Langlands is retired, 30th December, 1956.

The following officers are placed upon the Retired List (Southern Command) with permission to retain their rank and wear the prescribed uniform, 30th December, 1956:

Southern Command.—Major B. A. Baker and Captain (Honorary Major) T. P. Dawes.

Royal Australian Air Force.

Active Citizen Air Force.

Medical Branch.

No. 21 (City of Melbourne) Squadron.—Flight Lieutenant I. G. Nicol (0312763) is transferred from the Reserve, 11th August, 1956. Flight Lieutenant J. L. Colvin (015162) is transferred to the Reserve, 12th August, 1956.

No. 25 (City of Perth) Squadron.—Jack Sunderman (299561) is appointed to a commission, 13th April, 1956, with the rank of Flight Lieutenant. Flight Lieutenant J. McD. Quinlivan (051379) is transferred to the Reserve, 1st February, 1956.

Sydney University Squadron.—The following Pilot Officers are transferred to the Reserve: A. J. Reading (026836), G. M. Lambert (0211519), 20th June, 1956; R. J. Chapman (026761), 23rd July, 1956.

Air Force Reserve.

Medical Branch.

The following former officer is appointed to a commission, 16th June, 1956, with the rank of Squadron Leader: R. H. B. Sacks (036080).

The following are appointed to a commission with rank as indicated: (Flight Lieutenant (Temporary Squadron Leader)) Ernest John Egan (268061), 6th March, 1956; (Flight Lieutenant) Ben Totham Jervois Newland (287488), 23rd March, 1956; Rodney Francis McAuliffe (043069), 27th June, 1956; Donald Freebury King (257961), 28th June, 1956.

Flight Lieutenant E. L. Angliss (257824) is promoted to the temporary rank of Squadron Leader, 15th August, 1956.

The Royal Australasian College of Physicians.

VICTORIAN STATE COMMITTEE.

Lecture by Professor H. N. Robson.

The Victorian State Committee of The Royal Australasian College of Physicians has arranged for Professor H. N. Robson, Professor of Medicine, University of Adelaide, to deliver a lecture on "Mechanisms of Hæmostasis" in the lecture theatre of the Royal Australasian College of Surgeons, Spring Street, Melbourne, on Thursday, March 28, 1957, at 5.15 p.m. All members of the British Medical Association are invited to be present.

Medical Prizes.

BENGER PRIZES.

BENGER LABORATORIES, LIMITED, Holmes Chapel, Cheshire, are establishing a series of "Benger Prizes for Original Observations in General Practice". The prizes, expected to be sums of £250, £150 and £100 sterling, awarded annually, will be open to family doctors in any country, and will be awarded on the recommendations of the Awards Committee of the British College of General Practitioners. The ideas submitted may be concerned with the causation, diagnosis, treatment or prevention of any disease. It is hoped to publish all the entries in a book, which will be available to all, and which, it is hoped, will stimulate research workers, who will be expected to make acknowledgement to the doctor whose ideas are used. All profits from the sale of the book will go to the College of General Practitioners.

Entries must be received by July 31 in each year for the award in that year. The first award is to be made in 1957. Entries should be addressed to the Chairman, Awards Committee, College of General Practitioners, 54 Sloane Street, London, S.W.1, England. Further particulars may be obtained from Fison Chemicals (Pty.) Limited, Daking House, Rawson Place, Sydney.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE following additions and amendments have been made to the Register of Medical Practitioners for New South Wales in accordance with the *Medical Practitioners Act*, 1938-1955:

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (1) (a) of the Act: Allen, Eric Abbott, M.B., B.S., 1956 (Univ. Sydney); Chick, John Bertram, M.B., B.S., 1955 (Univ. Adelaide); Garlick, Frank Hender, M.B., B.S., 1951 (Univ. Queensland); McCann, William James, M.B., B.S., 1946 (Univ. Adelaide); Wassell, James Charles, M.B., B.S., 1954 (Univ. Queensland); Watkins, Rodney Alexander, M.B., 1956 (Univ. Queensland); Young, Marjorie Rose, M.B., Ch.B., 1951 (Univ. New Zealand).

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (1) (*b*) of the Act: Brooks, John Barritt, M.B., Ch.B., 1945 (Victoria Univ., Manchester); Gilderdale, Eric, M.B., Ch.B., 1945 (Univ. Edinburgh); Graham, Anthony Ismay, M.B., B.S., 1954 (Univ. London); Grassby, Geoffrey Claude, M.B., B.S., 1952 (Univ. London); Jolly, Terence Ginn, M.B., Ch.B., 1951 (Queen's Univ., Belfast); Mackenzie, John Lyle, M.B., Ch.B., 1944 (Univ. St. Andrews); Ryan, Douglas, M.R.C.S. (England), L.R.C.P. (London), 1944.

Registered medical practitioner who is required to complete twelve months' hospital service in accordance with the provisions of Section 17 (3) and is registered under Section 17 (1) (*a*) of the Act: Lucas, Eloise Mary Louisa, M.B., B.S., 1956 (Univ. Melbourne).

TASMANIA.

THE following have been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners: Parsons, Ronald Stanley, M.B., B.S., 1953 (Univ. Durham); Young, Peter Stewart, M.B., B.S., 1956 (Univ. Melbourne); Hodge, Donald Cleveland, M.B., B.S., 1956 (Univ. Melbourne); Smith, George Mackay, M.B., B.S., 1950 (Univ. Queensland); Bosanquet, Philip Walter George, M.B., B.S., 1955 (Univ. Sydney); Tarlinton, Michael John Kevin, M.B., B.S., 1955 (Univ. Sydney); Sundstrup, Bertel, M.B., B.S., 1955 (Univ. Sydney); Eley, John Russell, M.B., B.S., 1941 (Univ. Melbourne), D.T.M. & H. (London), 1953; Richards, Eric Dear, M.B., B.S., 1951 (Univ. Adelaide); Sloane, David Rae, M.B., B.S., 1956 (Univ. Sydney); Byatt, Noel Bradley, M.B., B.S., 1956 (Univ. Melbourne); Pianko, Isaac, M.B., B.S., 1956 (Univ. Melbourne).

The following additional diploma has been registered: Mathew, Randolph Yule, M.C.P.A., 1956.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED FEBRUARY 16, 1957.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	3(1)	2(1)	1(1)	..	..	..	..	..	6
Amœbiasis	..	1(1)	..	..	1	..	..	..	2
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	4(4)	9(9)	1(1)	..	..	..	6	1	21
Diphtheria	..	1(1)	..	..	..	1	..	..	2
Dysentery (Bacillary)	..	..	8(8)	..	..	..	..	..	8
Encephalitis	..	4(3)	..	..	..	..	..	..	4
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	1(1)	..	1	..	..	..	..	2
Infective Hepatitis	79(26)	35(18)	..	7(7)	2(1)	2(1)	..	2	127
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	10
Leptospirosis	..	..	10	..	..	..	..	..	1
Malaria	..	..	1(1)	..	..	..	..	..	2
Meningococcal Infection	..	2(1)	..	..	..	..	..	..	..
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	1(1)	..	..	..	..	..	..	..	1
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	4(1)	..	6	..	..	..	..	..	10
Puerperal Fever	2(2)	..	..	..	..	..	..	..	2
Rubella	..	44(20)	..	16(11)	..	..	..	..	60
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	4(3)	10(7)	2	3(1)	1(1)	1	..	..	21
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	1(1)	..	..	..	..	..	1
Trachoma	..	..	..	..	1	..	..	..	1
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	55(31)	20(16)	5(1)	7(3)	6(4)	2(1)	3	..	98
Typhoid Fever	1(1)	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	3	..	1(1)	..	..	..	4
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

Medical Appointments.

Dr. K. Shuley has been appointed Medical Officer of Health for the Municipality of Fingal, Tasmania.

Dr. A. K. Barrett has been appointed Honorary Associate Physician to the Allergy Department of the Royal Alexandra Hospital for Children, Sydney.

Dr. Colin William Phillips has been appointed a Quarantine Officer, Melbourne, in pursuance of the provisions of the *Quarantine Act*, 1908-1950.

Dr. Alan John Stubley has been appointed Senior Medical Officer, Mental Hygiene Branch, Department of Health, Victoria, pursuant to the provisions of Section 18 (2) of the *Mental Hygiene Authority Act*, 1950.

Dr. Ronald Munro Ford has been appointed Medical Superintendent at Morris Hospital, South Australia.

Dr. Robert Douglas has been appointed Medical Superintendent at Bedford Park Sanatorium, South Australia.

Dr. F. R. Hone has been appointed Honorary Consulting Physician at the Royal Adelaide Hospital.

Dr. A. E. Duxbury has been appointed Medical Research Officer at the Institute of Medical and Veterinary Science, Adelaide.

Dr. G. E. Summer has been appointed Pathology Registrar at the Institute of Medical and Veterinary Science, Adelaide.

Dr. R. B. Holland has been appointed Senior Medical Officer, New South Wales State Hospitals and Homes.

Dr. L. V. Perrett has been appointed Honorary Clinical Assistant to the Radiological Section at the Royal Adelaide Hospital.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Preswick, George, M.B., B.S., 1955 (Univ. Sydney), Clyde Street, Bateman's Bay, New South Wales.

Williams, Lorna Letitia, M.B., B.S., 1952 (Univ. Sydney), Box 51, Guyra, New South Wales.

The undermentioned have been elected as members of the New South Wales Branch of the British Medical Association: Appenzeller, Otto (provisionally registered), M.B., B.S., 1957 (Univ. Sydney); Bates, Edward Henry (provisionally registered), M.B., B.S., 1957 (Univ. Sydney); Beer, John Charles (provisionally registered), M.B., B.S., 1957 (Univ. Sydney); Billam, June Margaret (provisionally registered), M.B., B.S., 1957 (Univ. Sydney); Brown, John Maxwell (provisionally registered), M.B., B.S., 1957 (Univ. Sydney); Crakanthorp, John Churchill (provisionally registered), M.B., B.S., 1957 (Univ. Sydney); Evans, Hazel Jocelyn (provisionally registered), M.B., B.S., 1957 (Univ. Sydney); Hyslop, Raymond Stanley (provisionally registered), M.B., B.S., 1957 (Univ. Sydney); Lickiss, Jean Norelle (provisionally registered), M.B., B.S., 1957 (Univ. Sydney); McCredie, Kenneth John (provisionally registered), M.B., B.S., 1957 (Univ. Sydney); McMahon, Lynn Hamilton (provisionally registered), M.B., B.S., 1957 (Univ. Sydney); Mahony, George Grahame (provisionally registered), M.B., B.S., 1957 (Univ. Sydney); Mobbs, George Anthony (provisionally registered), M.B., B.S., 1957 (Univ. Sydney); Turner, James Edward (provisionally registered), M.B., B.S., 1957 (Univ. Sydney); Alexander, David Adolph, M.B., B.S., 1956 (Univ. Sydney); Burman, Ian Ronald, M.B., B.S., 1956 (Univ. Sydney); Child, Donald Stewart, M.B., B.S., 1956 (Univ. Sydney); Goddard, Kenneth Ernest, M.B., B.S., 1956 (Univ. Sydney); Kernot, Laurence William, M.B., B.S., 1956 (Univ. Sydney); Langford, Mae Vimiera Adelaide, M.B., B.S., 1956 (Univ. Sydney); Novotny, John, M.B., B.S., 1956 (Univ. Sydney); Shevill, Shirley, M.B., B.S., 1956 (Univ. Sydney); Tynan, Patrick James Felix, M.B., B.S., 1956 (Univ. Sydney); Wald, Louis, M.B., B.S., 1956 (Univ. Sydney); Baldwin, Douglas Bernard, M.B., B.S., 1955 (Univ. Sydney); Boon, Peter Eric, M.B., B.S., 1955 (Univ. Sydney); Bradley, Malcolm George, M.B., B.S., 1955 (Univ. Sydney); Hunt, John Francis, M.B., B.S., 1955 (Univ. Sydney); Maxwell, Donald Charles, M.B., B.S., 1955 (Univ. Sydney); Meyers, John, M.B., B.S., 1954 (Univ. Sydney); Oliver, Richard Dalley, M.B., B.S., 1954 (Univ. Sydney); O'Riordan, Richard Joseph, M.B., B.S., 1955 (Univ. Sydney); O'Sullivan, Eugene Peter, M.B., B.S., 1955 (Univ. Sydney); Pike, Geoffrey Howard, M.B., B.S., 1953 (Univ. Adelaide); Thew, Ian Phillip, M.B., B.S., 1954 (Univ. Sydney); Woolard, Thomas John, M.B., B.S., 1955 (Univ. Sydney); Lavrin, Leonide Mihailovitch, registered in accordance with the provisions of Section 17 (1) (c) of the *Medical Practitioners Act*, 1938-1955; Bobinskas, Mikas, registered in accordance with the provisions of Section 17 (1) (c) of the *Medical Practitions Act*, 1938-1955.

Deaths.

THE following deaths have been announced:

DENT.—Rowe Clyde Dent, on February 21, 1957, at Warwick, Queensland.

FINLEY.—Cecil Aubert Finley, on February 25, 1957, at Sydney.

Diary for the Month.

MARCH 12.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

MARCH 12.—New South Wales Branch, B.M.A.: Medical Politics Committee.

MARCH 16.—Western Australian Branch, B.M.A.: Annual General Meeting.

MARCH 16.—Tasmanian Branch, B.M.A.: Annual Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

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